University Of Alberta

Assessment of Outcomes from a Critical Pathway for Individuals Presenting to the Emergency Department with Symptomatic Atrial Fibrillation

By

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Abstract

There are Canadian and American evidence-based clinical guidelines for the treatment of Atrial Fibrillation (AF). However, outcomes of implementing a critical pathway based on these recommended guidelines have not been assessed. The purpose of this study was to assess the effects of initiating The Critical Pathway for Atrial Fibrillation for individuals with AF presenting to the Emergency Department (ED). A healthcare record review was undertaken. A convenience sample (n = 120) of two groups (Group A - Pre-Initiation of The Critical Pathway for Atrial Fibrillation; Group B – Post-Initiation of The Critical Pathway for Atrial Fibrillation) were assessed on the following outcomes: heart rate control, conversion of AF to sinus rhythm, and the initiation of an appropriate therapeutic anticoagulation regime. There was no statistically significant difference found on AF outcomes with the initiation of The Critical Pathway for Atrial Fibrillation. Heart rate control and rhythm conversion were similarly achieved, with improvement noted in follow-up care post-initiation of The Critical Pathway for Atrial Fibrillation.

Dedication

This thesis is dedicated to my son Nathan, whose independence and patience allowed me to fulfill this educational endeavour. To my husband, Leslie whose encouragement, strength and, love, inspired me during the final phases of this thesis. Also to my family, friends, and Grant MacEwan College colleagues whose support and friendship strengthened me throughout the years.

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Table Of Contents

Page

Chapter One

Introduction.....1

Purpose of the Study.....2

Definition of Terms.....3

Signification of the Study.....4

Chapter Two

Literature Review 6

Classification of Atrial Fibrillation 6

Mechanisms of Atrial Fibrillation.....7

Prevalence of Atrial Fibrillation 9

Etiology of Atrial Fibrillation 9

Outcomes of Atrial Fibrillation.....11

Diagnosis of Atrial Fibrillation.....13

Management of Atrial Fibrillation.....14

Chapter Three

Method.....22

Design.....22

Sample 23

Data Collection Procedure.....23

Data Analysis.....24

Ethical Considerations.....25

Chapter Four

Findings.....26

Admission Characteristics of Atrial Fibrillation
Patients Presenting to the Emergency Department.....26

Risk Factors for Atrial Fibrillation Patients Presenting
to the Emergency Department.....28

Admission Medications of Atrial Fibrillation Patients
Presenting to the Emergency Department.....32

Clinical Presentation of Atrial Fibrillation Patients
Presenting to the Emergency Department.....34
Diagnostics Requested for Atrial Fibrillation Patients
Presenting to the Emergency Department 35
Blood Work Requested for Atrial Fibrillation Patients
Presenting to the Emergency Department.....37
Heart Rate Control Pathway for Atrial Fibrillation
Patients Presenting to the Emergency Department.....38
Rhythm Control Pathway for Atrial Fibrillation
Patients Presenting to the Emergency Department 40
Anticoagulation for Individuals with Atrial Fibrillation Who Are
At High risk for Embolization Presenting to the
Emergency Department.....41
Follow-up Care Post Discharge of Atrial Fibrillation Patients
From the Emergency Department 43
Health Care Professionals' Satisfaction with
The Critical Pathway for Atrial Fibrillation 45

Chapter Five

Discussion of Findings 51
Diagnostic Approach for Atrial Fibrillation Patients
Presenting to the Emergency Department.....52
Heart Rate Control for Atrial Fibrillation Patients
Presenting to the Emergency Department.....56
Rhythm Control for Atrial Fibrillation Patients
Presenting to the Emergency Department 57
Appropriate Anticoagulation for Atrial Fibrillation Patients
Presenting to the Emergency Department.....61
Length of Stay of Atrial Fibrillation Patients
Presenting to the Emergency Department.....62
Emergency Department Health Care Professionals' Satisfaction
with The Critical Pathway for Atrial Fibrillation.....66

Limitations of the Study.....68
Implications for Current Practice 69
Conclusion.....72
References 75
Appendix A 89
Appendix B.....97
Appendix C.....100
Appendix D.....101

List of Tables

Page

Table 1

Admitting Characteristics of Atrial Fibrillation Patients Presenting to the Emergency Department 30

Table 2

Risk Factors for Atrial Fibrillation in Patients Presenting to the Emergency Department 31

Table 3

Medications of Atrial Fibrillation Patients Presenting to the Emergency Department 33

Table 4

Clinical Presentation of Atrial Fibrillation Patients Presenting to the Emergency Department.....35

Table 5

Diagnostics Requested for Atrial Fibrillation Patients Presenting to the Emergency Department 37

Table 6

Blood work Requested for Atrial Fibrillation Patients Presenting to the Emergency Department.....38

Table 7

Initiation of Rate Control Pathway for Atrial Fibrillation Patients Presenting to the Emergency Department.....40

Table 8

Initiation of Rhythm Control Pathway for Atrial Fibrillation Patients Presenting to the Emergency Department.....42

Table 9

Anti-coagulation Prior to DCC for Atrial Fibrillation Patients Presenting to the Emergency Department.....43

Table 10

Follow-up Care for Atrial Fibrillation Patients Presenting to the Emergency Department.....45

List of Figures

Page

Figure 1

The Critical Pathway for Atrial Fibrillation

Is Simple to Initiate.....46

Figure 2

The Critical Pathway for Atrial Fibrillation

Is Clear to Follow 47

Figure 3

The Critical Pathway for Atrial Fibrillation

Offers Complete Treatment.....48

Figure 4

The Critical Pathway for Atrial Fibrillation

Is Useful to Practice.....49

Figure 5

The Critical Pathway for Atrial Fibrillation

Is Helpful to Practice.....50

CHAPTER ONE

Introduction

Atrial fibrillation (AF) is a syndrome usually associated with underlying heart disease (Braunwald, Zipes, & Libby, 2001). The syndrome is described as disorganized and uncoordinated atrial activity without discrete "p" waves and with an irregularly irregular ventricular response resulting in the decline of effective atrial mechanical performance (Awtry & Loscalzo, 2001; Braunwald, Zipes, & Libby, 2001; Chakko & Myerburg, 1999; Cuddy & Connolly, 1996; Fuster & Rydén, 2001). Atrial fibrillation is the most common occurring dysrhythmia, projected at 0.4% in the general population (Ezekowitz & Levine, 1999; Fuster & Rydén, 2001, Hebar & Hueston, 2002). Although AF is the most common dysrhythmia presenting to Emergency Departments (ED), diagnostic and treatment approaches continue to vary and optimum treatment remains ambiguous (Khairy & Nattel, 2002; Savelieva & Camm, 2000). Current treatment guidelines are based on the individual's presenting clinical symptoms and hemodynamic state, as well as physician preference. Therefore, the ED is an ideal environment for the introduction of a critical pathway for promoting standardization of care, reducing complications and morbidity, and introducing potential cost-effective measures. The implementation of a critical pathway offers specific steps to guide healthcare professionals based on recommended evidence-based clinical guidelines (American Heart Association (AHA), 2001; Canadian Cardiovascular Society (CCS) Summary Statement, 1996). However, the outcomes of implementing a Critical Pathway for Atrial Fibrillation in the ED based on recommended clinical guidelines have not been assessed.

Purpose of the Study

A critical pathway may be a reliable, methodical, and proficient approach for the treatment of AF. The purpose of a critical pathway is to reflect evidence-based "best practice" (Woolf & George, 2000). Woolf and George (2000) suggested that even though research findings are disseminated, healthcare providers are often not motivated to change or alter treatment practices. Creating and implementing a critical pathway may ultimately aid in decreasing length of hospital stay, reducing costs, decreasing clinical uncertainty through standardized protocols for diagnosing and treating, and reducing unnecessary delays for the initiation of treatment (Bracken, 1997; Shevlin, Summers-Bean, Thomas, Whitney, Todd, & Ray, 2002; Robinson & Thomson, 2001; Wentworth & Atkinson, 1996; Woolf & George, 2000).

The purpose of this study was to develop, implement, and evaluate a Critical Pathway for Atrial Fibrillation (Appendix A) derived from published evidence-based clinical guidelines, for those individuals experiencing paroxysmal or persistent AF presenting to the ED.

The following hypotheses were tested:

1. There will be a more consistent and relevant diagnostic approach with the use of The Critical Pathway for Atrial Fibrillation than with current conventional approaches for individuals presenting to the Emergency Department with atrial fibrillation.
2. There will be better heart rate control achieved with the use of The Critical Pathway for Atrial Fibrillation than with current conventional treatment regimes for individuals presenting to the Emergency Department with atrial fibrillation.

3. There will be better conversion to sinus rhythm with the use of The Critical Pathway for Atrial Fibrillation than with current conventional treatment regimes for individuals presenting to the Emergency Department with atrial fibrillation.
4. There will be more initiation of appropriate anticoagulation with the use of The Critical Pathway for Atrial Fibrillation than with current conventional treatment regimes for individuals presenting to the Emergency Department with atrial fibrillation.
5. There will be a decreased length of time to see a physician in the Emergency Department with the use of The Critical Pathway for Atrial Fibrillation than with current conventional treatment regimes for individuals presenting to the Emergency Department with atrial fibrillation.
6. There will be greater health care professionals' satisfaction with the use of The Critical Pathway for Atrial Fibrillation than with current conventional treatment regimes for individuals presenting to the Emergency Department with atrial fibrillation.

Definition of Terms

Atrial fibrillation (AF) is defined as "a paroxysmal, persistent, or permanent supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with consequent deterioration of atrial mechanical function" (Fuster & Rydén, 2001, p. 4) as diagnosed by a 12 lead electrocardiogram (ECG).

A Critical Pathway for Atrial Fibrillation, are specific steps guiding healthcare professionals based on recommended Canadian and American clinical guidelines (AHA, 2001; CCS Summary Statement, 1996) (Appendix A).

Relevant diagnostic approach is defined as the requisition of appropriate diagnostics based on presenting clinical symptoms, existing co-morbidities, and current medication regimes according to American and Canadian guidelines (AHA, 2001; CCS Summary Statement, 1996) (Appendix A).

Heart rate control is defined as achieving a heart rate of less than 90 beats per minute (Fuster & Rydén, 2001; Gardner & Gilbert, 1996).

Conversion of AF is defined as the return to sinus rhythm and the presence of a "p" wave on a 12-lead ECG.

Therapeutic anticoagulation initiation is defined as appropriate risk stratification for embolization and subsequent initiation of appropriate anticoagulation to achieve an acceptable International Normalized Ratio (INR) range based on recommended Canadian and American guidelines (Albers, Dalen, Laupacis, Manning, Petersen, & Singer, 2001; Caro, Flegel, Oreguela, Kelley, Speckman, & Migliaccio-Walle, 1999; Connolly & Turpie, 1996; Ezekowitz & Levine, 1999; Lip & Li-Saw-Hee, 2000; Prystowsky, 2001).

Satisfaction of healthcare professionals is defined as the clarity, completeness, and ease of implementation of the Critical Pathway for AF as assessed by the Atrial Fibrillation Critical Pathway Questionnaire (Appendix B).

Significance of the Study

The importance and relevance of the study was reflected in the absence of existing critical pathways reflecting evidenced-based clinical guidelines for the treatment of AF. Although there are clinical practice guidelines available, there remains a lack of continuity in the diagnostic and treatment approach to the syndrome of AF in

current practice. The development and implementation of a critical pathway for AF may aid in determining drugs of choice for successful heart rate control, successful initiation of therapeutic anticoagulation regime, decreasing length of stay in ED, and thus potentially reducing costs. Following this study, The Critical Pathway for Atrial Fibrillation may be adopted within the Capital Health Region. This adoption may decrease clinical uncertainty through standardized protocols for diagnosing and treating, and reducing unnecessary delays for the initiation of treatment for AF in the health region.

CHAPTER TWO

Literature Review

Classification of Atrial Fibrillation

Fuster and Rydén (2001) classify primary AF as acute, chronic, paroxysmal, intermittent, constant, persistent, and/or permanent. Slower ventricular rates (< 60 beats/minute) imply disease of the AV node conduction system, while faster rates (> 180 beats/minute) may imply the existence of an accessory AV pathway (broad QRS, irregular rhythm), or an enhanced AV node conduction (narrow QRS complex) (Awtry & Loscalzo, 2001; Braunwald, Zipes, & Libby, 2001; Chakko & Myerburg, 1999; Cuddy & Connolly, 1996; Fuster & Rydén, 2001). The first presentation of AF requires identification as either a self-terminating or a persistent episode, and either symptomatic or asymptomatic (Khairy & Nattle, 2002). Recurrent AF is two or more episodes. Episodes identified as self-terminating are classified as paroxysmal and episodes identified as sustained are classified as persistent. The above terminology only reflects those cases of AF lasting longer than 30 seconds in duration and do not have a reversible cause. Secondary AF occurs from a variety of factors, including acute myocardial infarction, cardiac and non-cardiac surgery, pericarditis, myocarditis, hyperthyroidism, pulmonary embolism, pneumonia, or acute pulmonary disease. Once the treatment of the above factors is completed, often AF resolves, since it is not considered the primary problem (Alexander, Schlant, Fuster, O'Rourke, Roberts, & Sonnenblick, 1999; Fuster & Rydén, 2001). The last classification is known as "lone AF", which applies to individuals less than the age of 60 without underlying heart

and/or pulmonary disease and unknown etiology (Alexander, Schlant, Fuster, O'Rourke, Roberts, & Sonnenblick, 1999; Fuster & Rydén, 2001).

Mechanisms of Atrial Fibrillation

There are two main theories postulated for the mechanism of AF, arising from pathophysiological changes. The first theory originally proposed by Moe and Abildskov in 1959 and accepted by most, suggests a multiple-wavelet as the mechanism of reentrant AF (Fuster & Rydén, 2001; Prystowsky & Klein, 1994). These wavelets meander aimlessly throughout the atria advancing the creation of new wavelets. These new wavelets either collide with each other and are then respectively destroyed or continue to promote the creation of new wavelets; an endless occurrence (Braunwald, Zipes, & Libby, 2001). The creation of wavelets occurs based on the length of the refractory time, the mass of the atrium, and the conduction rate in various areas of the atria. The enlargement of the atria as seen in many AF patients is important in accommodating this first theory of mutual existence of multiple re-entrant wavelets (Fuster & Rydén, 2001).

The second theory suggests enhanced automaticity in one or more rapidly depolarizing foci promoting the development of AF (Fuster & Rydén, 2001; Khairy & Nattel, 2002, Kumagai, Khrestian & Waldo, 1997; Prystowsky & Klein, 1994). Automaticity refers to the ability of a cell to initiate an impulse unexpectedly without the need for initial stimulation (Braunwald, Zipes, & Libby, 2001). This stimulation may not reach a threshold to generate a cardiac action potential. However, the stimulation may reach the threshold potential and trigger another stimulation, thus causing self-perpetuating stimulations to occur (Braunwald, Zipes, & Libby, 2001).

Although the first two theories have been acknowledged over the years as the traditional understanding of the pathophysiology of AF mechanisms, there are currently new theories being postulated. The first of these theories suggests that the myocardial sleeve of the pulmonary vein may act as a foci, disseminating fibrillating waves to the atrium and therefore, initiating AF by acting as a repetitive focal discharge source (Zipes, 2003). Paroxysmal AF may have a central foci, and the pulmonary veins may perform a crucial task in the origin of this focal source (Everett & Olgin, 2004). Cheung, in 1981 demonstrated that the cardiac tissue surrounding the pulmonary veins could generate an action potential (as cited in Nattel, 2002). The rise of pulmonary veins as foci is thought to be due to atrial tachycardia remodeling, which may play an important role in triggering and maintaining AF (Zipes, 2003). "It is not clear whether the pulmonary veins have a role that is limited to actional potential generation, or whether (owing to the geometric arrangement of cardiac fibers around the veins or because of strands of poorly coupled cardiac tissue overlying vascular smooth muscle) they provide preferential zones for re-entry" (Nattel, 2002, p. 224). Focal triggers in the pulmonary veins may create short occurrences of self-sustaining AF, which in turn prompts other pulmonary vein sites to activate, thereby, reinitiating short runs of self-sustaining AF (Everett & Olgin, 2004). Recent findings have suggested that 94% of ectopic triggers for AF were found to arise from tissue around the pulmonary veins (Haissaguerre, Jais, Shah, Takahashi, Hocini, Quinious, et al., 1998). Ablation of these foci, prevented recurrence of AF in 62% of the patients, although less success has occurred in other studies (Haissaguerre, et al., 1998).

This abnormal automaticity may be a cause of delayed afterdepolarizations (DADs) arising from diseased tissue. The cause of DADs appear to be a result of a transient inward current that is deficient or absent under usual physiological states. These transient inward currents are the result of intracellular calcium overload resulting in accidental release of calcium from the sarcoplasmic reticulum which then activates chloride currents with an end result of a brief membrane depolarization. Pharmacological agents that reduce this sarcoplasmic calcium load (i.e., calcium channel antagonists, beta-adrenergic receptor blockers) or inhibit sarcoplasmic calcium release (thapsigargin, ryanodine, and/or cyclopiazonic acid) may suppress DADs (Zipes, 2003). Suppression of DADs may in turn suppress AF.

Prevalence of Atrial Fibrillation

Atrial fibrillation is one of the most common occurring arrhythmias, projected at 0.4% of the general population and increasing with age to 0.6% in individuals 80+ years old (Ezekowitz & Levine, 1999; Fuster & Rydén, 2001). According to the Government of Alberta (2000) there were 61 deaths attributed to AF for the year 1999, in ages 65 years and older. Age is the only identified independent risk factor for developing AF (Fuster & Rydén, 2001; Laupacis & Cuddy, 1996). Older individuals become at high risk for potentially developing AF, an independent predictor of mortality (Fuster & Rydén, 2001).

Etiology of Atrial Fibrillation

Although the triggers or causes of AF vary widely, they represent conditions advancing the occurrence of this dysrhythmia. These conditions may be grouped into four broad categories. The first category includes triggers that stretch the atria including

left ventricular hypertrophy with mitral valve disease, right ventricular hypertrophy with tricuspid valve disease, congenital heart defects, hypertension (HTN) and, coronary artery disease/acute myocardial infarction (MI) (Braunwald, Zipes, & Libby, 2001). These forms of heart disease may predispose an individual to ischemia in the atria and encourage the process of inflammation, a risk factor for the development of AF (Awtry & Loscalzo, 2001; Braunwald, Zipes, & Libby, 2001; Chakko & Myerburg, 1999; Cuddy & Connolly, 1996). The most direct cardiac dilemma of HTN is left ventricular hypertrophy which is related to left atrial size, a prominent predictor of AF (Crystal & Connolly, 2004). There is a greater incidence of AF with anterior MI versus inferior MI due to left atrial ischemia with potential complications of heart failure (HF), and excessive sympathetic stimulation (Awtry & Loscalzo, 2001; Braunwald, Zipes, & Libby, 2001; Cuddy & Connolly, 1996; Prystowsky, 2000).

The second category is scarring of the atria caused by triggers such as diabetes mellitus - biopsies revealed scarring of the left atrium in individuals with diabetes mellitus (Awtry & Loscalzo, 2001; Prystowsky, 2000). Another prominent risk factor for the occurrence of scarring is cardiac surgery. The incidence of AF increases in post-operative cardiac surgery individuals due to inflammation of the atria and increase in sympathetic tone following surgery (Asher, Miller, Grimm, Cosgrove & Chung, 1998; Bharucha & Kowey, 2000; Ellis, 1998; Mathew, Parks, Savine, Friedman, Koch, Mangano, & Browner, 1996; Moore & Wilkoff, 1991; Pagé & Pym, 1996).

The third category includes triggers, which infiltrate the atria. These factors include tumors involving the myocardium, angiosarcomas, amyloid heart disease, and sarcoidosis (Alexander, Schlant, Fuster, O'Rourke, Roberts, & Sonnenblick, 1999).

Persistent AF, which eventually leads to an increase in atrial dimensions, or a change in conduction of the atrium, is not only associated with underlying heart disease (Braunwald, Zipes, & Libby, 2001; Fuster & Rydén, 2001).

Thus, the final category includes all external or non-cardiac risk factors. These triggers include pericarditis, thyroid disease with concurrent angina and heart failure, non-cardiac surgery since bleeding, infection or an acid-base or electrolyte imbalance act as triggers post-operatively in the development of AF; and acute pulmonary disease which is associated with an increase in premature atrial contractions (PACs) acting as triggers for AF by lowering arterial oxygen tension or rising carbon dioxide levels (Awtry & Loscalzo, 2001; Braunwald, Zipes, & Libby, 2001; Cuddy & Connolly, 1996; Fuster & Rydén, 2001; Prystowsky, 2000). Also included in this final category are cor pulmonale also known as pulmonary HTN promoting hypoxia, acidosis, and hypercapnia (Braunwald, Zipes, & Libby, 2001; Chakko & Myerburg, 1999; Cuddy & Connolly, 1996). Others include drug formulation substitutions (substituting trade for generic brands with potential differences in bioavailability) which may decrease therapeutic drug levels of anti-arrhythmic agents, therefore promoting arrhythmia reoccurrence, proarrhythmia, or death (Reiffel, 2000); lone AF (idiopathic); and alcohol or "Holiday Heart Syndrome", due to an increase in catecholamine sensitivity (Chakko & Myerburg, 1999; Cuddy & Connolly, 1996; Fuster & Rydén, 2001; Prystowsky, 2000).

Outcomes of Atrial Fibrillation

The Framingham Study suggested individuals with a diagnosis of AF increase their mortality rate by a factor of 1.8 in comparison to those individuals without AF

(Laupacis & Cuddy, 1996). Thus, the mortality rate of both men and women with AF is nearly double that of individuals in normal sinus rhythm, and rises with the significance of the underlying heart disease (Fuster & Rydén, 2001). Cerebrovascular embolization occurs in 28% of those individuals with AF versus 7% in patients with sinus rhythm (Arnsdorf & Yip, 2000). These emboli may also circulate to other organs, including the kidneys, mesenteric circulation and the heart (Nattel, 2002). Furthermore, the introduction of AF in individuals with underlying heart disease worsens their prognosis since this dysrhythmia is an important risk factor for cerebral infarction (CCS Position Statement, 1996; Fuster & Rydén, 2001; Heart and Stroke Foundation of Canada, 1999). The addition of AF to underlying heart disease results in a 5-fold increase in cerebral infarction (Fuster & Rydén, 2001; Laupacis & Cuddy, 1996; Lin, Wolf, Kelly-Hayes, Beiser, Kase, Benhamin & D'Agostine, 1996; Wolf, Abbott, & Kannel, 1991). Adults over the age of 65 years with nonrheumatic heart disease and AF comprise 5-10% of all cerebral infarctions (CCS Summary Statement, 1996; Fuster & Rydén, 2001; Heart and Stroke Foundation of Canada, 1999). This worsens to a 17-fold increase in incidence of cerebral infarction in those individuals with rheumatic heart disease with AF (Fuster & Rydén, 2001). With such dire consequences, prevention of cerebral infarction needs to focus on lifestyle changes, control of HTN, and cholesterol levels, and appropriate atrial fibrillation management (Ezekowitz, Straus, Majumdar, & Finlay, 2003).

Other risk factors for stroke associated with AF include: mitral valve disease, prior stroke or transient ischemic attack (TIA), history of HTN, diabetes mellitus, and

moderate to severe left ventricular dysfunction/heart failure (Braunwald, Zipes, & Libby, 2001; Connolly & Turpie, 1996; Laupacis & Cuddy, 1996; Prystowksy, 2000).

Thus, the effects of AF on the circulatory system are seen as decreasing ventricular filling time, decreasing mean arterial pressure and increasing myocardial demand, resulting in impairment of cardiac output and promotion of atrial thrombus formation. Maintaining a sustained SR offers increasing maximal exercise capacity, decreasing left atrial dimensions, increasing left ventricular ejection fraction, and possibly improving quality of life – free of fatigue, exertional shortness of breath and/or angina (Braunwald, Zipes, & Libby, 2001; Fuster & Rydén, 2001; Gardner & Gilbert, 1996; Laupacis & Cuddy, 1996; Singh, 2000). Quality of life is also significantly impaired more in women than men, with the diagnosis of AF (Paquette, Roy, Talajic, Newman, Couturier, Yang, et al., 2000). Maintaining SR is vital, as it prevents electrical remodeling and associated atrial tachyarrrhythmias that may be present when AF becomes permanent (Khairy & Nattel, 2002).

Diagnosis of Atrial Fibrillation

Fatigue, angina, and exertional shortness of breath are often presenting symptoms of AF due to loss of atrial kick, rapid ventricular rate and a variable RR interval either at rest or during exertion (Awtry & Loscalzo, 2001; Prystoswky, 2000). However, the healthcare professional must also be aware there are patients who remain asymptomatic during AF episodes; reflected in a recent study that discovered 21% of patients did not have any obvious clinical presentations (Kerr, Boone, Connolly, Dorian, Green, Klein, et al., 1998). First line investigations include, a 12 lead ECG to confirm initial diagnosis and to provide information on underlying heart disease, serum

electrolytes (especially potassium, calcium and magnesium) to assist in identifying underlying imbalances as well as establishing a baseline, a complete blood count (CBC) to rule out anemia or evidence of an acute process, and finally a thyroid panel to identify hyperthyroidism (Fuster & Rydén, 2001; Gillis, Klein, & MacDonald, 1996; Khairy & Nattel, 2002).

Second line recommendations include chest x-ray to rule out critical situations necessitating emergency direct current (DC) cardioversion, such as flash pulmonary edema; International Normalized Ratio (INR), Partial Thromboplastin Time (PTT) for baseline values pre-anticoagulation; and cardiac enzymes to identify any imbalances requiring corrections (CCS Summary Statement, 1996; Fuster & Rydén, 2001). Other recommendations include, holter monitoring since a subgroup of patients develop AF during bradycardia and a low level exercise stress test (LLEST) to ascertain heart rate during exercise, and finally a transesophageal echocardiogram (TEE) to identify patients with AF > 48 hours in whom there is no evidence of a left atrial clot prior to cardioversion, and to identify valvular disease, left ventricular hypertrophy (LVH), and left atrial size (Fuster & Rydén, 20001; Gillis, Klein, & MacDonald, 1996; Prystowsky, 2000).

Management of Atrial Fibrillation

Primary prevention of AF looks at modifying potential risk factors. The first step is controlling underlying heart disease (ischemia, HF, and HTN). When managing HTN, angiotensin converting enzyme (ACE) inhibitors offer a 30% reduction in stroke versus other main stay antihypertensives (Yusuf, Sleight, Pogue, Davies, & Dagenais (2000). Other risk factors requiring correction are hypokalemia, hypomagnesemia, low

PO_2, hyperthyroidism, over use of stimulants, bradycardia, and prevention of trade versus generic drug substitution (Braunwald, Zipes, & Libby, 2001; Fuster & Rydén, 2001; Gardner & Gilbert, 1996; Prystowsky, 2000).

Secondary prevention deals with goals of therapy. Drug therapy for dysrhythmias, including AF, requires taking into account the patient's symptoms, severity of arrhythmia, underlying heart disease, and other patient characteristics including age, pre-existing pulmonary disease or liver dysfunction, especially with amiodarone regimes due to organ toxiticty. Maximizing patient's adherence to therapy is accomplished through convenient regimes, while minimizing risks for organ toxicity and proarrhythmia (Braunwald, Zipes, & Libby, 2001; Fuster & Rydén, 2001; Gardner & Gilbert, 1996; Newman, Gillis, Gilbert, & Dorian, 1996; Prystowsky, 2000).

Aggressiveness of management is reflected in the severity of presenting symptoms. The first goal of therapy is heart rate control preferably less than 80 - 90 beats per minute to ensure adequate cardiac output (Fuster & Rydén, 2001; Gardner & Gilbert, 1996; Hebbar & Hueston, 2002). Drug selection has to be based on underlying disease states. Heart rate control decreases symptoms associated with AF such as palpitations, fatigue, dyspnea, presyncope, syncope, and angina.

Although, amiodarone has been classified as a Class III antiarrhythmic agent, intravenously it has been shown to have alpha and beta blocking activity as well as calcium-channel blocking capabilities (Nattel, Talajie, Quantz, et al., 1987; Tsikouris & Cox, 2001). Amiodarone intravenously (IV) has been suggested as a potential effective pharmacological agent for both rate control and conversion [Class IIb recommendation (Appendix C), AHA, 2001] in individuals with preserved and impaired heart function

presenting with AF less than 48 hours duration, as well as effective in decreasing recurrent AF post DC cardioversion (AHA, 2001; Cotter, Blatt, Kaluski, et al., 1999; Galve & Ballester, et al., 1996). There have been many previous trials testing amiodarone IV in conversion of AF to SR, however, the results have been conflicting or trials have been small (Capucci, Lenzi, Boriani, et al., 1992; Cotter, Blatt, Kaluski, et al., 1999; Donovan, Power, Hockings, Dobb, & Lee, 1995; Hilleman & Spinler, 2002; Kumar, 1996; Tsikouris & Cox, 2001). However, recent trials have supported amiodarone as effective as other antiarrhythmic agents (Hilleman & Spinler, 2002), with a marked improvement in cardioversion rates after six to eight hours (Chevalier, et al., 2003). This agent (both oral and IV) is a potentially effective agent for treating AF due to its ability to increase atrial refractoriness, suppress atrial premature complexes, and control ventricular rate thus enhancing conversion, suppression, and prevention of refractory AF (AHA, 2001).

Oral administration of amiodarone in maintenance of SR has been found to be as superior to other agents (Roy, Talajic, Dorian, Connolly, Eisenber, Green, et al., 2000). Roy et al. (2000) discovered that amiodarone's ability to prevent recurrence of AF was significantly higher that either sotalol or propafenone (35% v. 63%, $p < 0.0001$).

Advantages of using amiodarone include low cardiac toxicity, lower proarrhythmia risk in comparison with other antiarrhythmic agents, comparable or better efficacy rate than other antiarrhythmic agents, and an acceptable drug for those individuals with impaired heart function (Hilleman & Spinler, 2002; Roy, Talajic, Dorian, et al., 2000; Tsikouris & Cox, 2001). Disadvantages include a long-half life, large volume of distribution, highly lipophilic, poor bioavailability, increase in costs

(for agent as well as screening for high risk non-cardiac organ toxicity primarily pulmonary, thyroid, opthamological, gastrointestinal, and hepatic), and commonly reported adverse effects of infusion phlebitis with IV form, bradycardia and finally hypotension (Hilleman & Spinler, 2002; Tsikouris & Cox, 2001).

However, in individuals with preserved heart function [> 40% Ejection Fraction (EF)] where amiodarone is not available, or there is a contraindication for its use, one of the following will assist in heart rate control: calcium channel blockers or beta-blockers, both Class I recommendations (Appendix C) (AHA, 2001; Fuster & Rydén, 2001; Gardner & Gilbert, 1996; Prystowsky, 2000; Singh, 2000). However, in those individuals with impaired heart function (< 40% EF), the use of only one of the following is suggested, digoxin (Class Ib, Appendix C), or diltiazem (Class IIb, Appendix C) (AHA, 2001; Fuster & Rydén, 2001; Gardner & Gilbert, 1996; Prystowsky, 2000; Singh, 2000).

Conversion to sinus rhythm (SR) is the second goal of therapy. Spontaneous conversion occurs in 0 – 48% of the population (Talajic, MacDonald, & Nattel, 1996; Van Gelder, Tuinenburg, Schoonderwoerd, Tieleman, & Crijns, 2000). The deciding factor, according to these authors, for optimal spontaneous conversion is duration of AF less than 72 hours. Converting AF to SR relieves symptoms, as well as decreases the incidence of thromboembolism, prevents cardiomyopathy, circumvents electrical and anatomical remodeling, and restores effective atrial performance (Braunwald, Zipes, & Libby, 2001) and may improve quality of life (Hagens, Ranchor, Van Sonderen, Bosker, Kamp, Tijssen, Kingma, Harry, Crijns, & Van Gelder, 2004). Electrical or direct current (DC) is one means of converting AF to SR. Indications for immediate

electrical cardioversion include: active ischemia, significant hypotension with signs of hypoperfusion, rapid ventricular response (usually greater than 150 beats per minute) and acute pulmonary distress (AHA, 2001; Fuster & Rydén, 2001; Khairy & Nattel, 2002; Talajic, MacDonald, & Nattel, 1996; Van Gelder, Tuinenburg, Schoonderwoerd, Tieleman, & Crijns, 2000). Although termination of AF occurs in 70 – 90% of cases, AF reoccurs in up to 50 – 75%, following initial conversion to SR. This may necessitate a pharmacological agent(s) to maintain SR (Prystowsky, 2000; Talajic, MacDonald, & Nattel, 1996).

Direct current cardioversion is most effective with AF less than 72 hours, possibly due to anatomical and electrical remodeling of the atria during chronic AF (AHA, 2001; Fuster & Rydén, 2001; Gardner & Gilbert, 1996; Prystowsky, 2000). Though DC cardioversion may lead to stunning in recent AF, and hibernation in chronic AF, it is still effective; 75% success rate if the arrhythmia is present for less than 48 hours (Talajic, MacDonald, & Nattel, 1996). Atrial thrombus formation is thought to occur over a 2-week period. Since embolization occurs in 1.2% of patients who are not anticoagulated, anticoagulation of 3 weeks duration is recommended prior to DC cardioversion for AF greater than 48 hours to prevent adhesion of atrial thrombus to the atrial wall. A small study (165 subjects) comparing a biphasic cardioversion approach to a standard monophasic approach discovered a 94% versus 79% successful conversion rate from AF to SR (Mittal, Ayati, & Stein, et al., 2000). Also an anterior-posterior approach was also discovered to be more successful than the previous anterior-anterior positioning. The anterior-posterior approach offers an effective current flow through critical areas of the heart since low-energy shocks may

be delivered for effective cardioversion (Mittal, Ayati, & Stein, et al., 2000). Post cardioversion anticoagulation for a 4-week "minimum", due to a high risk of a thromboembolic event when reverting back to SR, is recommended (AHA, 2001; Braunwald, Zipes, & Libby, 2001; Fuster & Rydén, 2001; Prystowsky, 2000; Talajic, MacDonald, & Nattel, 1996).

The use of pharmacological agents is the second means of converting AF to sinus rhythm. This form of conversion is recommended for individuals in AF who do not present with serious signs and symptoms of tachycardia, and heart rate is less than 150 beats per minute (AHA, 2001; Fuster & Rydén, 2001; Prystowsky, 2001; Talajic, MacDonald, & Nattel, 1996). However, drug selection is also based on heart function. If the duration of AF is less than 48 hours and there is preserved heart function (> 40% EF) one of the following may be used, amiodarone (Class IIa), ibutide (Class IIa), flecainide (Class IIa), propafenone (Class IIa), and procainamide (Class IIa) (AHA, 2001; Fuster & Rydén, 2001; Prystowsky, 2000; Talajic, MacDonald, & Nattel, 1996). These authors suggested if the duration of AF is less than 48 hours and there is impaired heart function (< 40% EF); consider DC cardioversion or amiodarone (Class IIb). However, the introduction of antiarrhythmic drugs can also lead to life-threatening rhythm disorders – proarrhythmias (i.e., torsades de pointe) increasing an individual's morality (Nattel, 2002).

The third goal of therapy is minimizing the risk of embolization. The risk of embolization is greatest soon after the onset of AF or with conversion back to sinus rhythm (Braunwald, Zipes, & Libby, 2001; Fuster & Rydén, 2001; Heart and Stroke Foundation of Canada, 1999; Talajic, MacDonald & Nattel, 1996), and usually occurs

within the first 72 hours (King, Dickerson, & Sack, 2002). In those individuals presenting with AF greater than 48 hours and also requiring urgent DC cardioversion, the choice of anticoagulation has been unfractionated heparin (UFH) (AHA, 2001; Connolly & Turpie, 1996; Fuster & Rydén, 2001; Prystowsky, 2000). However, recent literature has supported the use of low molecular weight heparin (LMWH) as an alternative to UFH for short-term anticoagulation use (Camm, 2001; Harenberg, Weuster, Pfitzer, Dempfle, Stehle, Kubler, & Schlier, 1993; Murray, Deitcher, Shah, Jasper, Bashir, Grimm, & Klein, 2001; Raskob, 1995; Shulman, 2000). The advantages of LMWH include, not requiring intravenous administration, hospitalization, laboratory monitoring and dose adjustment, or healthcare professional administration (Camm, 2001; Harenberg, Weuster, Pfitzer, Dempfle, Stehle, Kubler, & Schlier, 1993; Murray, Deitcher, Shah, Jasper, Bashir, Grimm, & Klein, 2001; Raskob, 1995; Shulman, 2000). If long-term anticoagulation is necessary, administration of warfarin reduces the incidence of stroke by 68% as compared to 21% reduction with aspirin, even though warfarin use is under-utilized in all populations, but notably in the young (<55 years) and in the elderly (> 85 years) (Connolly & Turpie, 1996; Lip & Li-Saw-Hee, 2000; The Heart Organization, 1999). Long-term anticoagulation is highly recommended in individuals who have not had successful cardioversion and who are not ideal candidates for either repeat DCC or pharmacological conversion (King, Dickerson, & Sack, 2002).

The therapeutic range for INR is recommended between 2.0 – 3.0 (Connolly & Turpie, 1996; Ezekowitz & Levine, 1999; Lip & Li-Saw-Hee, 2000; Prystowsky, 2001). However, antithrombotic therapy requires risk stratification prior to initiation to prevent complications (Arnsdorf & Podrid, 2000; Connolly & Turpie, 1996; Fuster &

Rydén, 2001; Lip & Li-Saw-Hee, 2000). Categories for risk stratification are identified as high risk, moderate risk, and low risk. There is a high risk for developing a thrombus (6% per year) in those individuals with HTN, diabetes, history of a prior stroke or TIA, coronary artery disease or HF (Fuster & Rydén, 2001; Hauser, 2000). If warfarin is not contraindicated, the INR goal is 2.5. However if individuals are older than 75 years of age, they may be treated with the same range as above or a lower therapeutic goal of INR 2.0 (Connolly & Turpie, 1996; Fuster & Rydén, 2001; Hauser, 2000). The second category, are those individuals with a moderate risk (2% per year) for developing a thrombus. These are individuals who are older than 65 years of age but have no high risk factors, as mentioned previously. Prevention of an embolic event occurs through the use of either warfarin (INR range as above) or enteric coated acetylsalicylic acid (ECASA) 325 mg per day (Fuster & Rydén, 2001; Hauser, 2000). The low risk group (1% per year for developing a thrombus) is individuals less than 65 years of age, who do not have any high risk factors. The only recommendation for prevention of a CVA due to AF is with the use of ECASA 325 mg per day (Fuster & Rydén, 2001; Hauser, 2000). However, determination of bleeding risk and relative contraindications must be calculated prior to the administration of any antithrombotic. Contraindications include: an inability to control prothrombin time, dementia, malignancy, previous serious bleeding problems, and uncontrolled serious HTN (Chakko & Myerburg, 1999; Connolly & Turpie, 1996; Hauser, 2000; Phillips, 1998; Singh, 2000).

CHAPTER THREE

Method

Design

The purpose of this study was to develop, implement, and evaluate The Critical Pathway for Atrial Fibrillation (Appendix A) derived from published evidence-based clinical guidelines for those individuals presenting with paroxysmal or persistent atrial fibrillation (AF) to an Emergency Department. A comparative design (Group A: Pre-Initiation of The Critical Pathway for Atrial Fibrillation; Group B: Post-Initiation of The Critical Pathway for Atrial Fibrillation) utilizing a retrospective health record review to examine the relationship between the use of a critical pathway and clinical outcomes, in individuals presenting to the Grey Nuns Community Hospital (GNCH) ED with AF. The clinical outcomes that were assessed were diagnostic approach, heart rate control, conversion of AF to SR, initiation of therapeutic anticoagulation, length of stay in the ED, and healthcare professionals' adherence and satisfaction.

The initial phase of the study included an extensive review of the literature to ascertain best evidence-based practice for the diagnosis and treatment of AF, leading to the creation of The Critical Pathway for Atrial Fibrillation. Three cardiologists specializing in cardiac electrophysiology, four cardiologists at the GNCH, and also three ED physicians at the GNCH critiqued earlier drafts. Recommendations from these critiques were incorporated into the final Critical Pathway for Atrial Fibrillation. The final step prior to implementation of the critical pathway was approval from the Patient Services Section, Cardiac Sciences Program Council, and Capital Health Region,

whose recommendations were also included in the final Critical Pathway for Atrial Fibrillation.

Sample

Healthcare records of all individuals over the age of 18 presenting to the GNCH ED with the diagnosis of atrial fibrillation were eligible for inclusion in the study. The GNCH is a 214 bed facility. This hospital serves a population base of 83,601 individuals in the southeast portion of Edmonton, Alberta (City of Edmonton, November 2001). According to Tadra Boulton, Public Affairs Specialist at the GNCH, emergency admissions for the year 2000-2001 were greater than 60,000 individuals and are expected to increase. It is anticipated that approximately 10-15 individuals per month are admitted to the GNCH ED with a diagnosis of AF.

Group A included individuals in the retrospective arm of the study (Pre-Initiation of The Critical Pathway for Atrial Fibrillation) and Group B included individuals in the prospective arm of the study (Post-Initiation of The Critical Pathway for Atrial Fibrillation). For Group A, 60 health records of individuals who presented to the GNCH ED from January to August 2002 were reviewed. For Group B, 60 health records of individuals who presented to the GNCH ED from November 2002 to July 2003 were reviewed.

Data Collection Procedure

Approval was obtained from the Patient Services Section, Cardiac Sciences Program Council, Capital Health Region and the Nursing Care Manager and Medical Director of the GNCH ED for the implementation of The Critical Pathway for Atrial Fibrillation. An in-service on The Critical Pathway for Atrial Fibrillation was given for

the medical and nursing staff working at the GNCH ED prior to the implementation of The Critical Pathway for Atrial Fibrillation in November 2002.

A list of health records with the diagnosis of AF/dysrhythmia was obtained from the Health Information Analyst GNCH, on a monthly basis. This list was then submitted to the Health Care Records Department at the GNCH to retrieve the identified health records. Health records were then reviewed and The Critical Pathway for Atrial Fibrillation outcome data were recorded on the data collection tool (Appendix D).

Upon completion of enrollment for Group B, ED healthcare professionals were mailed a letter of introduction and the Atrial Fibrillation Critical Pathway Questionnaire (Appendix B) to assess the clarity, completeness, and ease of implementation of The Critical Pathway for Atrial Fibrillation. A list of healthcare professionals was obtained through the ED Nursing Care Manager and Chief Medical Officer for the GNCH and a package was mailed to them via GNCH. Healthcare professionals had a month to return the questionnaire in the self-addressed stamped envelope. A follow-up reminder was sent at this time.

Data Analysis

Descriptive statistics including measures of central tendency (median, mean, standard deviation, and range) were used. A Chi-square test or a *t* test, where appropriate, was completed to analyze the differences between groups and the outcome variables under examination following the implementation of The Critical Pathway for Atrial Fibrillation. The level of significance for all tests was $p \leq 0.05$.

Ethical Considerations

Ethical approval was obtained from the Health Research Ethics Board and Capital Health Region prior to commencement of the study. Support for the study was obtained from both the Nursing Manager and Medical Director of the GNCH ED and the Patient Services Section, Cardiac Sciences Program Council, Capital Health Region. Data were organized by code numbers and have been kept in a locked filing cabinet. Analysis and publication of the data will not identify individuals within this study.

CHAPTER FOUR

Findings

The purpose of the study was to develop, implement, and evaluate The Critical Pathway for Atrial Fibrillation (Appendix A) derived from published evidence-based clinical guidelines for those individuals presenting with paroxysmal or persistent atrial fibrillation (AF) to the Grey Nuns Community Hospital Emergency Department (ED). The Critical Pathway for Atrial Fibrillation reflected standardized protocols for diagnostics, rate and rhythm control, as well as follow up care for patients presenting to the ED with AF. Descriptive statistics including measures of central tendency (median, mean, standard deviation, and range) were used. A Chi-square test or a *t* test, where appropriate, was implemented to analyze the differences between groups on the outcome variables under examination following the implementation of The Critical Pathway for Atrial Fibrillation. The level of significance was $p \leq 0.05$.

Admission Characteristics of Atrial Fibrillation Patients Presenting to the Emergency Department

Health records of all individuals over the age of 18 presenting to the Grey Nuns Community Hospital Emergency Department with the diagnosis of AF were eligible for inclusion in the study. A total of 120 individuals were eligible and their health care records were reviewed. Participants in the study ranged in age from 25 to 92 years (Median = 68.5, M = 66.25, SD = 16.32). There was no significant difference between the Pre-Initiation of The Critical Pathway for Atrial Fibrillation (Group A) (M = 65.78, SD = 15.88) and the Post-Initiation of The Critical Pathway for Atrial

Fibrillation (Group B) (M = 66.72, SD = 16.87) with respect to age ($p = 0.76$) (Table 1).

Prior to arrival in the ED, patients' length of time in AF, ranged from less than one hour to a maximum of 672 hours (11 patient's length of time was not stated), with no significant difference between Group A and Group B (p = 0.07) (Table 1). Group A had mean length of time of 14.19 hours (SD = 24.06), whereas Group B had a mean length of time of 47.71 hours (SD = 135.44) ($p = 0.07$).

The time to see an emergency physician for all AF patients ranged from 4 to 281 minutes, with a mean of 47.34 minutes and a median of 34.00 minutes. The introduction of The Critical Pathway for Atrial Fibrillation was expected to decrease length of time to see a physician in the ED. However, there was no significant difference between Group A (M = 39.83 minutes, SD = 32.06) and Group B (M = 54.54 minutes, SD = 50.60) with respect to initial contact with an emergency physician ($p = 0.97$) (Table 1).

Comparisons were made between the groups on baseline vital signs (temperature, heart rate, blood pressure, and oxygen saturation).Temperatures were taken for 100 of the 120 eligible patients with a range from 34.1° – 38.3° Celsius. Group A had a mean temperature of 36.47° Celsius (SD = 0.74) and Group B had a mean temperature of 36.42° Celsius (SD = 36.42) ($p = 0.70$).The initial heart rate on presentation of the patient with AF to the ED was taken on all patients, with a range of 52 to181 beats/minute. The mean heart rate for Group A was 119 beats/minute (SD = 31.25) and for Group B was 119 beats/minute (SD = 32.13). Again, there was

no statistically significant difference found between groups with respect to heart rate ($p = 0.95$).

Systolic blood pressure (BP) was measured for all AF patients with a range from 72 to 250 mm Hg. The Pre-Initiation of The Critical Pathway for Atrial Fibrillation (Group A) had a mean systolic BP of 140 mm Hg (SD = 28.48) and the Post-Initiation of The Critical Pathway for Atrial Fibrillation group (Group B) had a mean systolic BP of 131 mm Hg (SD = 22.87). There was no statistically significant difference between groups with respect to systolic BP ($p = 0.88$) (Table 1). There also was no statistically significant difference between groups in terms of diastolic BP, as Group A had a mean diastolic BP of 89 mm Hg (SD = 17.51) with an equivalent value of 89 mm Hg (SD = 18.88) in Group B ($p = 0.88$) (Table 1).

The last admitting characteristic monitored in both groups was oxygen saturation (O_2 sat) either on room air or with supplemental oxygen (Table 1). This characteristic was taken on 102 of the total 120 AF patients presenting to the ED, with a range from 83 to 100% O_2 saturation. The Pre-Initiation of The Critical Pathway for Atrial Fibrillation (Group A) had a mean O_2 sat of 96% (SD = 3.05) and The Post-Initiation of The Critical Pathway for Atrial Fibrillation (Group B) had a similar mean O_2 sat of 95% (SD = 2.50) ($p = 0.07$).

Risk Factors for Atrial Fibrillation Patients Presenting to the Emergency Department

There are many risk factors identified in the literature that may predispose an individual to develop AF (Table 2). These factors include cardiac, non-cardiac, and other precipitants (i.e., alcohol). Overall, there were no statistically significant differences between Group A and B, on risk factors for AF (Table 2). One identified

risk factor from the literature was gender, with 60% of males and 40% of females presenting with AF to the ED in Group A, and with 56.67% of males and 43.44% of females in Group B presenting with AF ($p = 0.85$). Another risk factor was hypertension, where 25% of Group A presented with hypertension, when compared to 36.7% of Group B ($p = 0.24$). A third risk factor identified in the literature was myocardial infarction (MI). Results were comparable between groups, with 10 individuals in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group having experienced a MI compared to 9 individuals in the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group. Thyroid disease, a fourth risk factor, had 9 individuals presenting to the ED with this disease in Group A, compared to 5 in Group B ($p = 0.39$).

Table 1

Admitting Characteristics of Atrial Fibrillation Patients Presenting to the Emergency Department

	Pre-Initiation of Critical Pathway (n = 60)		Post Initiation of Critical Pathway (n = 60)		
Characteristic	**Mean**	**SD**	**Mean**	**SD**	***p***
Age	65.78	15.88	66.72	16.87	0.76
Length of time in AF prior to arrival in ED (hours)	14.19	24.06	47.71	135.44	0.07
Time to see Physician (minutes)	39.83	32.06	54.54	50.60	0.97
Temperature	36.47	0.74	36.42	0.63	0.70
Heart Rate (beats/minute)	119.48	31.25	119.13	32.13	0.95
Systolic BP	140.05	28.48	131.95	22.87	0.88
Diastolic BP	89.55	17.51	89.37	18.87	0.96
O_2 Saturation	96.36	3.05	95.33	2.50	0.07

$p < .05$, independent sample *t*-test

BP = blood pressure

Table 2

Risk Factors for Atrial Fibrillation in Patients Presenting to the Emergency Department

Risk Factors	Pre-Initiation of Critical Pathway (n=60) Frequency (Percent)	Post-Initiation of Critical Pathway (n=60) Frequency (Percent)	*p*
Gender			0.85
Male	36(60.00)	34(56.67)	
Female	24(40.00)	26(43.33)	
HTN			0.24
Yes	15(25.00)	22(36.67)	
No	45(75.00)	38(63.33)	
Valvular Heart Disease			1.00
Yes	2(0.33)	2(0.33)	
No	58(96.67)	58(96.67)	
CAD			0.68
Yes	2(3.33)	4(6.77)	
No	58(96.67)	56(93.33)	
MI			1.00
Yes	9(15.00)	10(17.00)	
No	51(85.00)	50(83.33)	
CHF			1.00
Yes	6(10.00)	6(10.00)	
No	54(90.00)	54(90.00)	
CABG			0.62
Yes	3(5.00)	1(1.67)	
No	57(95.00)	59(98.33)	
Family History			0.37
Yes	4(6.67)	1(1.67)	
No	56(93.33)	59(98.33)	
Recent Surgery			1.00
Yes	1(1.67)	0(0.00)	
No	59(98.33)	60(100.00)	
Thyroid			0.39
Yes	9(15.00)	5(8.33)	
No	51(85.00)	55(91.67)	
Diabetes			0.79
Yes	7(11.67)	9(15.00)	
No	53(88.33)	51(85.00)	
Acute Pulmonary Disease			0.24
Yes	3(5.00)	0(0.00)	
No	57(95.00)	60(100.00)	
Substance Abuse			1.00
Yes	2(3.33)	3(5.00)	
No	58(96.67)	57(95.00)	
Infection			1.00
Yes	1(1.67)	2(3.33)	
No	59(98.33)	58(96.67)	
Acid-base			0.24
Yes	3(5.00)	0(0.00)	
No	57(95.00)	60(100.00)	

$p < .05$, Chi-Square, Fisher's Exact Test

Admission Medications of Atrial Fibrillation Patients Presenting to the Emergency Department

Many AF patients presenting to the ED have co-morbidities that are treated with appropriate prescribed medication. No statistically significant difference was noted between prescribed medications on admission to the ED between groups (Table 3). Medications to treat hypertension and angina were frequently ordered prior to admission in both groups. Although both groups were prescribed ACE-inhibitors prior to admission, Group A had 14 individuals on this medication as compared to 21 individuals in Group B ($p = 0.23$). Again with respect to calcium channel blockers, Group A had 5 individuals contrasted to Group B with 9 individuals ($p = 0.40$) taking calcium channel blockers. The last classification, beta-blockers, were ordered similarly between Group A (21 individuals) and Group B (19 individuals) ($p = 0.85$).

An important medication identified in the literature to decrease those at high risk for embolization was either an anticoagulation and/or aspirin regime. Of the AF individuals in Group A, 30% were taking an anticoagulant as compared to 23% in Group B ($p = 0.54$). In the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group, aspirin was taken by 25% of AF individuals contrasted to 18% in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group ($p = 0.51$).

Table 3

Medications of Atrial Fibrillation Patients Presenting to Emergency Department

	Pre-Initiation of Critical Pathway (n=60)	Post-Initiation of Critical Pathway (n=60)	
	Frequency(Percent)	Frequency(Percent)	*p*
Medications			
Ace-Inhibitor			0.23
Yes	14(23.33)	21(35.00)	
No	46(76.67)	39(65.00)	
Antiarrhythmic			1.00
Yes	8(13.33)	8(13.33)	
No	52(86.67)	52(86.67)	
Anticoagulant			0.54
Yes	18(30.00)	14(23.33)	
No	42(70.00)	46(76.67)	
Aspirin			0.51
Yes	15(25.00)	11(18.33)	
No	45(75.00)	49(81.67)	
Beta-blocker			0.85
Yes	21(35.00)	19(31.67)	
No	39(65.00)	41(68.33)	
Calcium Channel Blocker			0.40
Yes	5(8.33)	9(15.00)	
No	55(91.67)	51(85.00)	
Lipid Lowering Agents			0.18
Yes	9(15.00)	16(26.67)	
No	51(85.00)	44(73.33)	
Oral Hypoglycemics/Insulin			0.56
Yes	5(8.33)	8(13.33)	
No	55(91.67)	52(86.67)	
Digoxin			0.76
Yes	7(11.67)	5(8.33)	
No	53(88.33)	55(91.67)	
Diuretic			0.49
Yes	10(16.67)	14(23.33)	
No	50(83.33)	46(76.67)	
Synthroid			0.56
Yes	10(16.67)	5(8.33)	
No	50(83.33)	55(91.67)	
Other Meds			1.00
Yes	40(66.67)	39(65.00)	
No	20(33.33)	21(35.00)	
No Meds			0.30
Yes	6(10.00)	11(18.33)	
No	54(90.00)	49(81.67)	

$p < .05$, Chi-Square, Fisher's Exact Test

Clinical Presentation of Atrial Fibrillation Patients Presenting to the Emergency Department

There were no differences between Group A and Group B with classification of AF into categories. Group A had 47 individuals presenting with AF < 48 hours, compared to 49 individuals in Group B (p = 0.82). Similar numbers were found between the two groups, with 13 individuals in Group A presenting with AF > 48 hours and 11 individuals in Group B (p = 0.82).

With respect to the first occurrence of AF among individuals presenting to the ED, there was no significant difference between groups, Pre-Initiation of Critical Pathway for Atrial Fibrillation (23.33%) and Post-Initiation of Critical Pathway for Atrial Fibrillation (25.00%) ($p = 0.89$) (Table 4). However, there was a significant difference between groups with respect to the number of occurrences of AF ($p = 0.04$). The Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group had more individuals having >11 previous episodes of AF (n = 4) than the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group (n = 2), while the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group had more individuals with intermittent AF (n = 8) than the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group (n = 0).

Table 4

Clinical Presentation of Atrial Fibrillation Patients Presenting to the Emergency Department

	Pre-Initiation of Critical Pathway (n=60)	Post-Initiation of Critical Pathway (n=60)	
	Frequency(Percent)	**Frequency(Percent)**	***p***
Occurrence			
< 48 Hours	47(78.33)	49(81.67)	0.82
> 48 Hours	13(21.66)	11(18.33)	0.82
First Occurrence	14(23.33)	15(25.00)	0.89
Occurrences			**0.04**
1-5	20(33.33)	20(33.33)	
6-10	2(3.33)	1(1.67)	
>11	4(6.67)	2(3.33)	
Chronic	10(16.67)	8(13.33)	
Intermittent	0(0.00)	8(13.33)	
Not stated	11(18.33)	5(8.33)	

$p > .05$, Pearson Chi-Square

Diagnostics Requested for Atrial Fibrillation Patients Presenting to the Emergency Department

Standard diagnostics for AF outlined in evidenced-based guidelines and reflected in The Critical Pathway for Atrial Fibrillation included: telemetry, vital signs with oxygen (O_2) saturation, a 12 lead electrocardiogram (ECG), and chest x-ray (Table 5). With the introduction of The Critical Pathway for Atrial Fibrillation, it was expected that there would be a more consistent and relevant diagnostic approach to the treatment of AF. However, there were no significant differences in diagnostic approach found between groups.

The most frequent diagnostic test ordered in both groups was a 12 lead ECG. However, only 95% of individuals in The Pre-Initiation of The Critical Pathway for

Atrial Fibrillation Group received a telemetry recording and/or a 12 lead ECG. The Post-Initiation of The Critical Pathway for Atrial Fibrillation Group, had similar results with 96.67% of individuals receiving a telemetry recording and all individuals receiving a 12 lead ECG.

There was no significant difference between groups when requested diagnostics were reviewed. As previously mentioned, vital signs (VS) determine the hemodynamic status of an individual with AF, and should direct further initiation of diagnostics and/or treatment. There were no significant differences between the ordered baseline vital signs between groups. In Group A, vital signs were ordered on 86.67% of the AF individuals, as compared to Group B, with 95% of all AF individuals with recorded vital signs ($p = 0.20$).

The least ordered diagnostic test was a chest x-ray. There were no significant differences between groups with requests for a chest x-ray. In The Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group, 13 individuals had a chest x-ray, compared to 15 individuals in The Post-Initiation of The Critical Pathway for Atrial Fibrillation Group ($p = 0.83$).

Table 5

Diagnostics Requested for Atrial Fibrillation Patients Presenting to the Emergency Department

	Pre-Initiation of Critical Pathway (n=60)	Post-Initiation of Critical Pathway (n=60)	
	Frequency(Percent)	**Frequency(Percent)**	***p***
Diagnostic			
Telemetry	57(95.00)	58(96.67)	1.00
VS & 0_2 saturation	52(86.67)	57(95.00)	0.20
12 lead ECG	57(95.00)	60(100.00)	0.24
Chest x-ray	18(30.00)	16(26.67)	0.84
Other	13(21.67)	15(25.00)	0.83

$p < .05$, Chi-Square, Fisher's Exact Test
VS = vital signs
O_2 = oxygen saturation
ECG - electrocardiogram

Blood Work Requested for Atrial Fibrillation Patients Presenting to the Emergency Department

The blood work requested in The Critical Pathway for Atrial Fibrillation as initial work-up for individuals who presented with AF included: a CBC with differential, electrolytes, an anticoagulation status, and screening for potential cardiac and thyroid emergencies (Table 6). These standard crucial blood tests for the possible detection of precipitants should help determine AF triggers and, therefore, appropriate treatment.

There were no significant differences found between groups. A CBC with differential and electrolytes were the two most frequently ordered blood tests, with greater than 70% of all individuals with AF having these tests. Although there were no statistically significant differences between groups, more glucose, PT, PTT, and

troponin tests were ordered in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group.

Table 6

Blood Work Requested for Atrial Fibrillation Patients Presenting to the Emergency Department

	Pre-Initiation of Critical Pathway (n=60)	**Post-Initiation of Critical Pathway** (n=60)	
Blood Work	**Frequency(Percent)**	**Frequency(Percent)**	***p***
CBC (differential)	44(73.33)	44(73.33)	1.00
Sodium	45(75.00)	42(70.00)	0.68
Potassium	45(75.00)	42(70.00)	0.68
Chloride	45(75.00)	42(70.00)	0.68
CO_2	45(75.00)	42(70.00)	0.68
Creatinine	44(73.33)	41(68.33)	0.68
Blood urea	42(70.00)	38(63.33)	0.56
Magnesium	18(30.00)	16(26.67)	0.84
Calcium	14(23.33)	13(21.67)	1.00
Glucose	29(48.33)	40(66.67)	0.06
PT(INR)	10(16.67)	20(33.33)	0.06
PTT	5(8.33)	11(18.33)	0.18
CK	20(33.33)	16(26.67)	0.55
CKMB	18(30.00)	15(25.00)	0.68
Troponin	12(20.00)	18(30.00)	0.29
TSH	11(18.33)	7(11.67)	0.44

$p < .05$, Chi-Square, Fisher's Exact Test

Heart Rate Control Pathway for Atrial Fibrillation Patients Presenting to the Emergency Department

Rate control is the initial choice of treatment for those individuals in The Critical Pathway for Atrial Fibrillation who are hemodynamically stable. The ideal medications for pharmacologically controlling heart rate as suggested by the evidenced-based clinical

guidelines included calcium-channel blockers, beta-blockers, and inotropic drugs (Table 7).

The pathway was initiated with equal frequency between the groups; ED physicians treated 19 in the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group and 20 individuals in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group. Medications prescribed included recommended heart rate control regimes from The Critical Pathway for Atrial Fibrillation. These intravenous medications were ranked identically between the groups, with metoprolol being the most commonly ordered medication, followed by digoxin, diltiazem, and verapamil.

There were no differences between the achievements of rate control of AF between groups. Rate control was successful in 15 of the 19 individuals in the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group, and 11 of the 20 individuals in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group. Rate control was considered successful if the individual's heart rate was less than 90 beats/minute.

Table 7

Initiation of Rate Control Pathway for Atrial Fibrillation Patients Presenting to Emergency Department

	Pre-Initiation of Critical Pathway	Post-Initiation of Critical Pathway	
	Frequency(Percent)	Frequency(Percent)	*p*
Rate Control Initiated	19(31.67)	20(33.33)	1.00
Diltiazem IV	4(6.67)	2(3.33)	0.68
Metoprolol IV	13(21.67)	9(15.00)	0.48
Verampamil IV	2(3.33)	2(3.33)	1.00
Digoxin	4(6.67)	10(16.67)	0.15
Rate Control Achieved			0.51
Yes	15(25.00)	11(18.33)	
No	45(75.00)	49(81.67)	

$p < .05$, Chi-Square, Fisher's Exact Test

Rhythm Control Pathway for Atrial Fibrillation Patients Presenting to the Emergency Department

The rhythm control pathway is the second choice for treatment following stabilization of the patient in The Critical Pathway for Atrial Fibrillation. There was an expectation that individuals presenting to the ED with AF would have higher conversion rates from AF to sinus rhythm (SR) with the introduction of The Critical Pathway for Atrial Fibrillation. However, findings indicated there was not a significant difference in overall conversion rates between groups.

There was no significant difference in choosing this option between groups (Table 8). This pathway was initiated for 31 individuals with AF in the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group, compared to 33 individuals in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group. Both the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group (30.00%) and the Post-

Initiation of The Critical Pathway Group (36.67%) chose intravenous procainamide as their pharmacological conversion agent (Table 8).

Direct current cardioversion (DCC) was another option in the rhythm control pathway and was chosen equally between the two groups. The success of DCC was 55.00% in the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group, and 53.33% in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group in converting AF to SR. However, permanent success of DCC was only 71.67 % in the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group, and only 68.33% in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group upon discharge from the Emergency Department (Table 8).

Anticoagulation for Individuals with Atrial Fibrillation Who are at High Risk for Embolization Presenting to the Emergency Department

The Critical Pathway for Atrial Fibrillation clearly outlined individuals at risk and appropriate corresponding anticoagulation regimes. However, this treatment was not utilized frequently in either group (Table 9). Only two individuals in the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group received anti-coagulation prior to direct current cardioversion (DCC); one patient received Heparin intravenously and the second received unfractionated heparin – enoxaparin. In the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group, three patients received warfarin for a minimum of four weeks prior to an elective DCC as an out-patient in the Emergency Department (Table 9).

Table 8

Initiation of Rhythm Control Pathway for Atrial Fibrillation Patients Presenting to the Emergency Department

	Pre-Initiation of Critical Pathway	Post-Initiation of Critical Pathway	
Rhythm Control	**Frequency(Percent)**	**Frequency(Percent)**	***p***
Rhythm Control Initiated	31(51.67)	33(55.00)	1.00
Anti-arrhythmic Ordered	21(35.00)	17(28.33)	0.56
Flecainide po	1(1.67)	0 (0.00)	1.00
Procainamide IV	18(30.00)	22(36.67)	0.56
Propafenone po	6(10.00)	11(18.33)	0.11
DC Cardioversion Sedation			0.89
Propofol	17(28.33)	18(30.00)	
Other	3(5.00)	2(3.33)	
Joules			0.27
100	4(6.67)	0(0.00)	
200	6(10.00)	9(15.00)	
300	5(8.33)	3(5.00)	
360	3(5.00)	2(3.33)	
Biphasic	2(3.33)	5(8.33)	
Able to Convert			0.78
Yes	33(55.00)	32(53.33)	
No	17(28.33)	18(30.00)	
Spontaneously	10(16.67)	9(15.00)	
Remain in SR			0.58
Yes	43(71.67)	41(68.33)	
No	17(28.33)	18(30.00)	
Not stated	0(0.00)	1(1.67)	

$p < .05$ Chi-Square. Fisher's Exact Test
ED = emergency department
po = oral route
IV = intravenous route
SR = sinus rhythm

Table 9

Anti-coagulation Prior to DCC for Atrial Fibrillation Patients at High Risk for Embolization Presenting to the Emergency Department

	Pre-Initiation of Critical Pathway	Post-Initiation of Critical Pathway
Anti-coagulation	**Frequency(Percent)**	**Frequency(Percent)**
Heparin IV	1(1.67)	0(0.00)
Bolus	1(1.67)	0(0.00)
Infusion	1(1.67)	0(0.00)
Unfractionated Heparin	1(1.67)	0(0.00)
Enoxaparin	1(1.67)	0(0.00)
****Oral Dose**	0(0.00)	3(5.00)

$p < .05$, Chi-Square, Fisher's Exact Test

DCC = direct current cardioversion

AF = Atrial Fibrillation

** (Minimum of 4 weeks prior to elective DC cardioversion)

Follow-up Care Post Discharge of Atrial Fibrillation Patients From the Emergency Department

An essential component identified in The Critical Pathway for Atrial Fibrillation was follow-up care. Follow-up care consisted of all care documented by ED physicians upon discharge. This care consisted of cardiology consults, admission to an in-patient unit in the hospital, booking elective procedures as out-patients (TEE and/or DCC), and follow-up with a family physician in the community (Table 10). There was a statistically significant difference between groups with respect to the ordering of follow-up care for AF individuals. In The Pre-Initiation of The Critical Pathway for

Atrial Fibrillation Group, 40% received follow-up care, while 60% in The Post-Initiation of The Critical Pathway for Atrial Fibrillation Group, received follow-up care.

A cardiology consult was not only the most frequent component of follow-up care requested by ED physicians, but was ordered equally (23.33%) in both groups. More TEEs and follow-up with family physicians were ordered in The Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group. Individuals with AF were admitted equally to an in-patient unit in the health care region. An elective DCC as an out-patient was infrequently ordered by ED physicians.

The Critical Pathway for Atrial Fibrillation clearly outlined individuals at risk and appropriate corresponding anticoagulation regimes. However, this treatment was not utilized frequently in either group, and there were no significant differences between groups (Table 10).

Table 10

Follow-up Care for Atrial Fibrillation Patients Discharged from the Emergency Department

	Pre-Initiation of Protocol	Post-Initiation of Protocol	
	Frequency(Percent)	**Frequency(Percent)**	***p***
Follow-up Care	24(40.00)	36(60.00)	**0.04**
Categories of Care			
Cardiology Consult	14(23.33)	14(23.33)	1.00
Admit to hospital	5(8.33)	5(8.33)	1.00
Book TEE	9(15.00)	3(5.00)	0.13
Book outpatient DCC	2 (3.33)	1(1.67)	1.00
Family physician	8(13.33)	4(6.67)	0.36
Initiation of Warfarin	12(20.00)	14(23.33)	0.83

$p < .05$, Chi-Square, Fisher's Exact Test
TEE = transesophageal echocardiogram
DCC = direct current cardioversion

Healthcare Professionals' Satisfaction with The Critical Pathway for Atrial Fibrillation

The development and implementation of The Critical Pathway for Atrial Fibrillation derived from evidenced-based clinical guidelines was expected to enhance current conventional AF treatment regimes in the ED. The guidelines were developed for simplicity, clarity, and completeness, for initiation by the ED physician. The expectation of the initiation of The Critical Pathway for Atrial Fibrillation in the ED, was that it would increase health care professionals' satisfaction.

A survey was distributed to health care professionals (physicians and nurses) employed in the ED following the completion of enrollment. The questionnaire consisted of five questions ranked on a Likert scale from strongly disagree to strongly

agree, with an option to write further comments. The response rate from the physicians was 60 percent (9 out of 15 physicians), however, there was no response from the nursing staff, even after multiple attempts.

The first question asked was, *"Is The Critical Pathway for Atrial Fibrillation simple to initiate?"* Overall, only 33% of the ED physicians responded that the pathway was simple to initiate. No further comments or critiques were given to explain the responses.

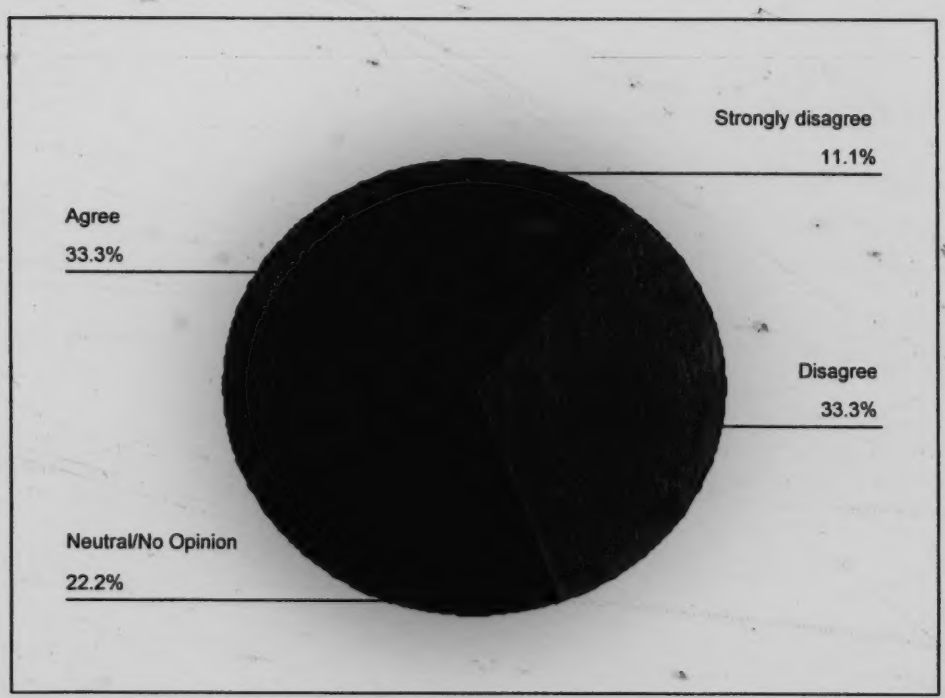

Figure 1. The Critical Pathway for Atrial Fibrillation is Simple to Initiate

"Is The Critical Pathway for Atrial Fibrillation clear to follow?", was the second question polled. Over 55 % of the respondents agreed the pathway was clear to follow, while 22.22% were either neutral or had no opinion. However, further constructive feedback and/or critiques were lacking in the comment section offered to respondents.

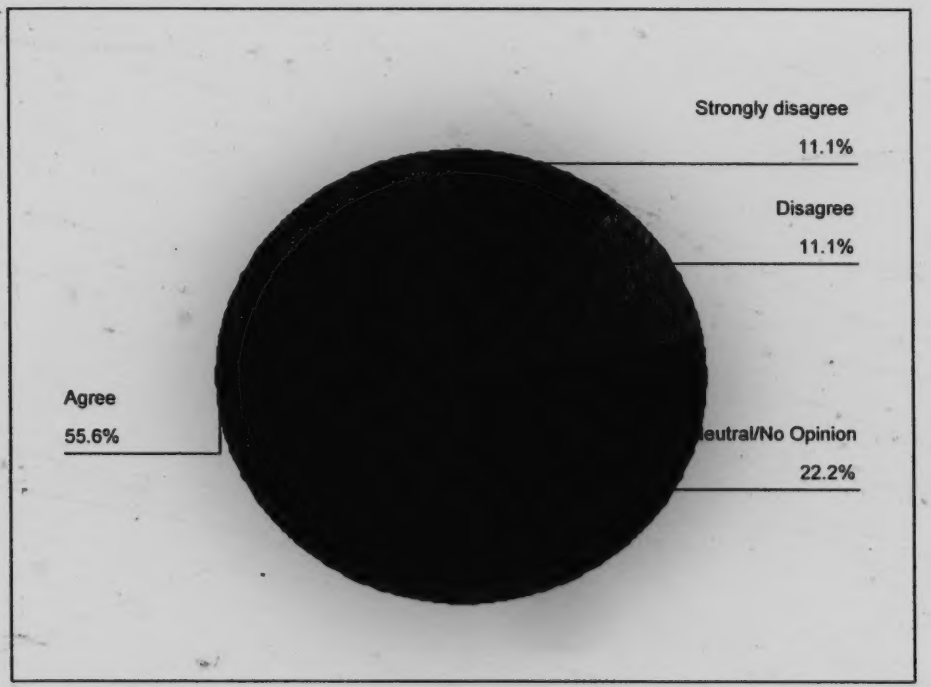

Figure 2. The Critical Pathway for Atrial Fibrillation is Clear to Follow

The third question asked was, *"Is The Critical Pathway for Atrial Fibrillation complete in the treatments suggested?"*. Again over 55% of the respondents agreed the treatments suggested were complete based on existing guidelines. However, 22.2% of the respondents were neutral or had no opinion and 22.2% responded that the treatments suggested were not complete. Comments provided were, "Don't need PTT or INR as baseline", and "Where is IV procainamide for unsuccessful DC cardioversion stable pt? Is the study sponsored by amiodarone makers?"

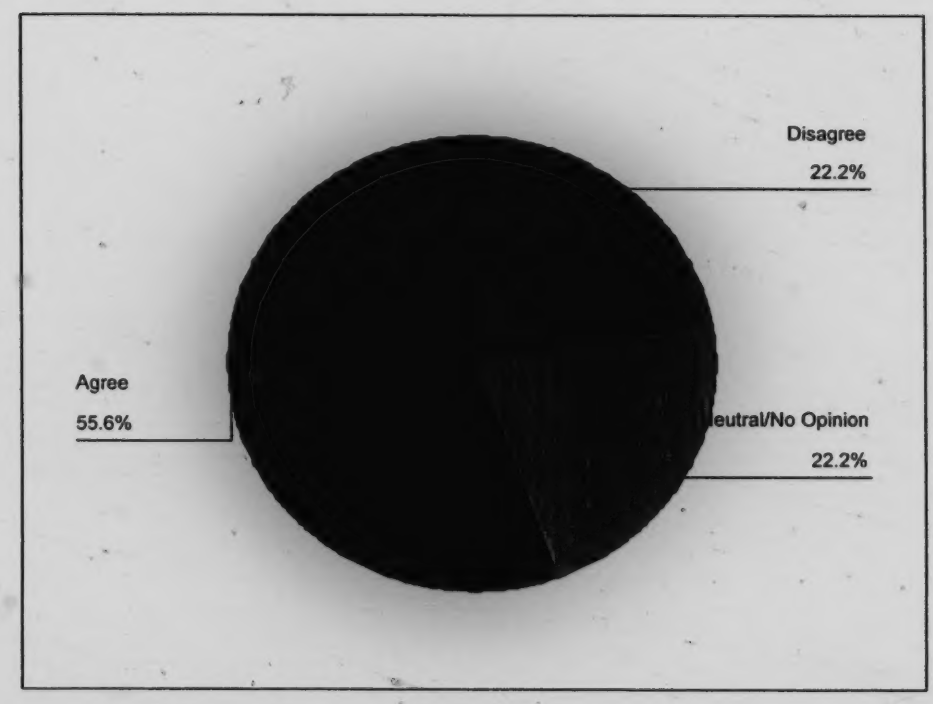

Figure 3. The Critical Pathway for Atrial Fibrillation Offers Complete Treatment

"*Is The Critical Pathway for Atrial Fibrillation useful to your practice?*", was the fourth question on the survey. The overall results were less than positive with 44.4 percent of the respondents staying neutral/no opinion, with the remaining responses replying that they either disagreed (44.4 percent) or strongly disagreed (11.1 percent).

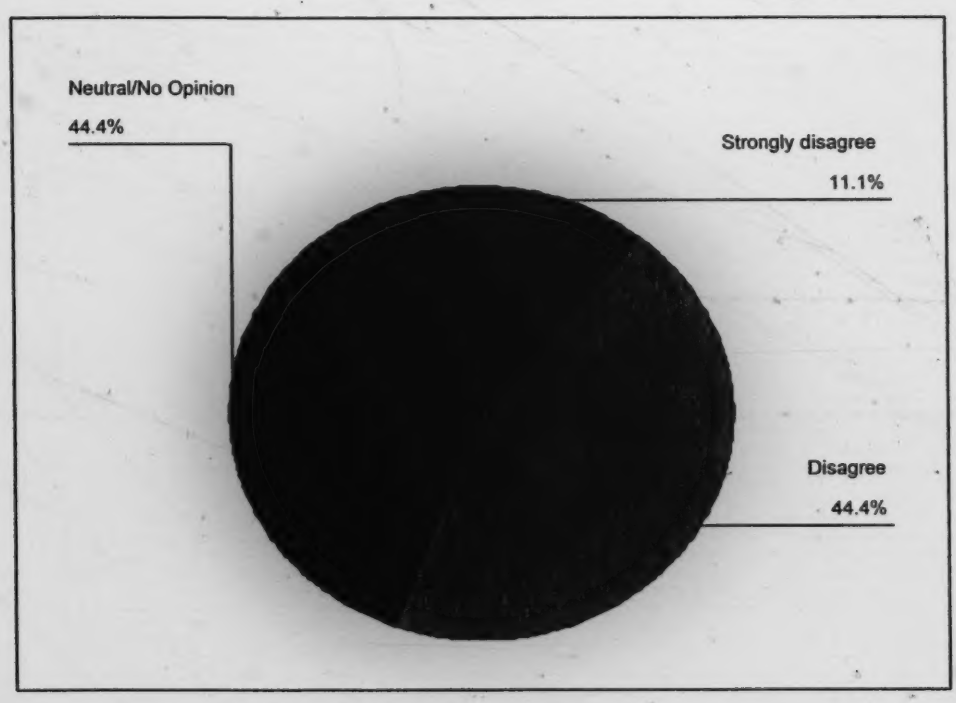

Figure 4. The Critical Pathway for Atrial Fibrillation is Useful to Practice

The final question was, "*Is The Critical Pathway for Atrial Fibrillation helpful in providing care?*", was once again less than positive. An overwhelming number of responses (77.8 percent) remained neutral or had no opinion on this particular question on the survey, with the remaining (approximately 22.2 percent) stating they disagreed with the critical pathway being helpful to their practice.

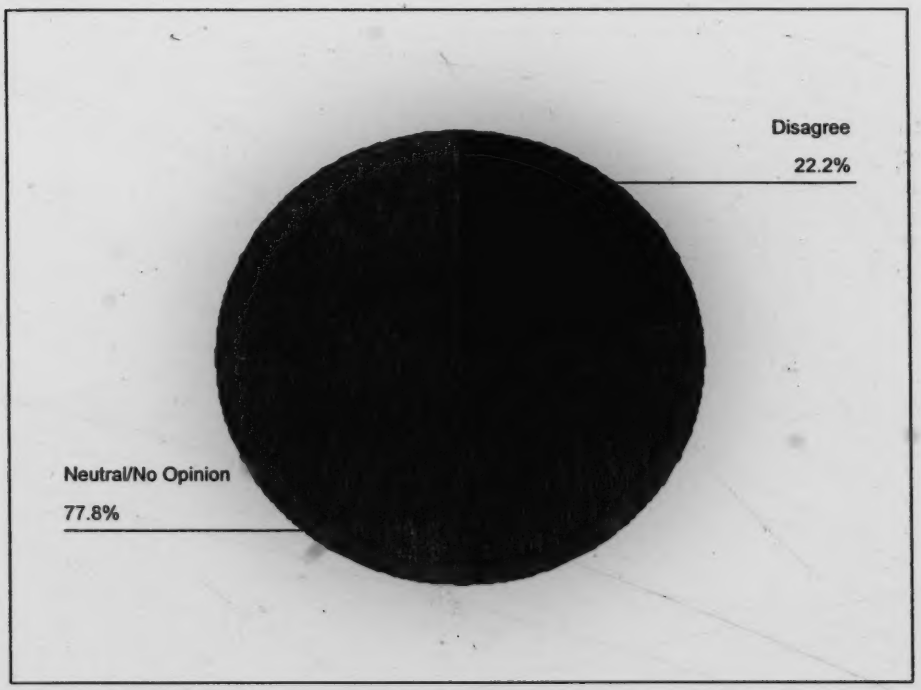

Figure 5. The Critical Pathway for Atrial Fibrillation is Helpful to Practice

Additional comments from one individual and which did not fit the content of the above questions included, "Wrong induction dose of propofol: Try 1mg (1000ug)/kg IV push. Biphasic not available at most places."

CHAPTER FIVE

Discussion of Findings

The purpose of the study was to develop, implement, and evaluate The Critical Pathway for Atrial Fibrillation (AF), derived from published evidence-based clinical guidelines, in an Emergency Department (ED). A comparative design (Group A: Pre-Initiation of the Critical Pathway; Group B: Post-Initiation of The Critical Pathway for Atrial Fibrillation) was used to examine the relationship between the use of a critical pathway and clinical outcomes, through the use of a retrospective health record review, of individuals presenting to the Grey Nuns Community Hospital (GNCH) ED with AF.

The initial phase of the study included an extensive review of the literature to ascertain best evidence-based practice for the diagnosis and treatment of AF, leading to the creation of The Critical Pathway for Atrial Fibrillation (Appendix A). The Critical Pathway for Atrial Fibrillation reflected standardized protocols for diagnostics, rate and rhythm control, as well as follow up care for patients presenting to the Emergency Department with atrial fibrillation. Descriptive statistics including measures of central tendency (median, mean, standard deviation, and range) were used. A Chi-square test or a *t* test, where appropriate, was implemented to analyze the differences between groups and the outcome variables under examination following the implementation of The Critical Pathway for Atrial Fibrillation. The level of significance was $p \leq 0.05$.

Both the Pre-Initiation of The Critical Pathway for Atrial Fibrillation group, and the Post-Initiation of The Critical Pathway for Atrial Fibrillation group were comparable, with no significant differences with respect to the majority of admission characteristics. Admission characteristics included: age, baseline vital signs, gender,

risk factors for the development of AF, admission medications, length of time in hours of AF prior to presentation to the ED, and length of time to see a physician.

Rationale for the lack of overall significance in the study may include the following: this particular ED not only has four cardiologists on service, but also participates in many cardiovascular research studies; this institution offers weekly cardiology and medical rounds to the health care team to broaden their knowledge base; and many individual physicians in the ED may ensure their practice is derived from existing published evidenced-based clinical guidelines.

Of significance was in the clinical presentation of AF patients presenting to the ED. Atrial fibrillation patients were assigned to either the < or > 48 hours category prior to initiation of the appropriate clinical pathway. Similar numbers were found between the two groups. However, there was a statistically significant difference ($p = 0.04$) in number of occurrences between the two groups. The Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group had more individuals presenting with >11 episodes which does impact the course of care. There is a potential of a 25% yearly conversion rate from intermittent AF into a chronic AF state in those patients with underlying coronary disease and increasing age (Crystal & Connolly, 2004). Therefore, those individuals of advancing age with frequent intermittent presentations require a thorough cardiac workup and appropriate follow-up.

Diagnostic Approach for Atrial Fibrillation Patients Presenting to the Emergency Department

With the introduction of The Critical Pathway for Atrial Fibrillation, it was expected that there would be a more consistent and relevant diagnostic approach to the

treatment of AF. No difference was found between the conventional diagnostic approach and the approach recommended by The Critical Pathway for Atrial Fibrillation, for individuals presenting to the ED with AF. No statistically significant differences were noted between Group A and Group B, with regards to standard diagnostic and/or blood tests (12 lead ECG, CBC with differential, electrolytes, anticoagulation status, screening for cardiac, thyroid and substance abuse, and radiographic studies) recommended by The Critical Pathway for Atrial Fibrillation. Differences between the two groups were absent, perhaps because diagnostic tests were ordered appropriately for AF individuals (with AF < or > 48 hours) presenting to the ED by the physicians based on presenting symptoms, past medical history, and evidenced-based practice guidelines (American Heart Association (AHA), 2001; Canadian Cardiovascular Society (CCS) Summary Statement, 1996) both Pre and Post-Initiation of The Critical Pathway for Atrial Fibrillation.

However, more PT/INR tests were ordered on patients post-initiation of The Critical Pathway for Atrial Fibrillation ($p = 0.06$). It is important for a baseline value of PT/INR to be ordered to determine therapeutic effects of existing prescribed anticoagulation medication, as well as a baseline for treating those individuals who are at high risk for a stroke (Taylor, Cohen, & Embrahim, 2001). Since AF is an underlying cause of embolic strokes, it is vital to decrease the risk of atrial thrombi occurrence, since the risk of atrial thrombi occurrence from AF dramatically increases with age, and is especially high 23.5% in elderly patients aged 80 to 85 years (Albers, Dalen, Paupacis, Manning, Petersen, & Singer, 2001). Although the mean age of all patients (N = 120) was 66.25 years, 22 individuals were older than 80 years of age (18.33%).

Although there were no statistically significant differences noted between the two groups with respect to PT/INR ($p = 0.06$), the lack of significance does not suggest best practice for the prevention of AF strokes occurred in either the Pre-Initiation of The Critical Pathway for Atrial Fibrillation or the Post-Initiation of The Critical Pathway for Atrial Fibrillation Groups. Best practice, as outlined by evidenced-based clinical guidelines and reflected in The Critical Pathway for Atrial Fibrillation, suggests a baseline PT/INR as an appropriate diagnostic screening test.

In the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group, there were 7 individuals over the age of 80 who were at high risk for the development of a thrombus based on their presenting risk factors (HTN, Diabetes, CAD, and/or, HF). These individuals were not on an anticoagulation or ECASA regime prior to presentation to the ED, and only one individual received a baseline PT/INR as an appropriate diagnostic test based on recommendations from The Critical Pathway for Atrial Fibrillation during their ED stay. These 7 individuals were discharged from the ED without an order for appropriate anti-embolic medication to decrease their risk for thrombus development. Three of these individuals (2 with chronic intermittent AF and one presenting with 2 episodes within a 5 month period) had a DCC without post-discharge anticoagulation. Furthermore, there were 4 individuals (45, 57, 58, and, 65 years of age) with moderate to high risk for thrombus development who had either multiple episodes or a long standing history of AF who were not on an anticoagulation regime prior to presentation. These individuals also did not receive a baseline PT/INR and only one received a prescription for warfarin upon discharge, although all 4 individuals had cardioversion.

In the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group, 3 individuals over the age of 80 presented with co-morbidities, which placed them at high risk for thrombus development, with only 2 individuals receiving a baseline PT/INR prior to pharmacological conversion. Also there were 3 individuals (44, 65, and 76 years of age) with moderate to high risk for thrombus development who did present on admission to the ED on warfarin/coumadin, but did not receive a PT/INR to determine therapeutic effectiveness, prior to DCC.

Appropriate anticoagulation for those individuals at high risk for thrombus development and those individuals post DCC was recommended by The Critical Pathway for Atrial Fibrillation derived from evidenced-based clinical guidelines. However, these guidelines were not always followed (as previously demonstrated) as best practice in either of the groups. The majority of AF strokes occur within the first 72 hours after conversion (pharmacologic or DCC) (Albers, Dalen, Laupacis, Manning, Petersen, & Singer, 2001). This lack of prescribing anticoagulation regimes for patients at high risk for thrombus development reflects the current pattern of under-utilization in the elderly by health care professionals, exposing them to an increase risk of a CVA (Caro, Flegel, Orejuela, Kelly, Speckman, & Migliaccio-Walle, 1999; Cohen, Almoznino-Sarafian, Alon, Gorelik, Koopfer, & Chacashvily, et al., 2003). This lack of prescribing is often based on physician's preference due to fear of cerebrovascular hemorrhages due to dementia, falls, and lack of medication compliance.

Heart Rate Control for Atrial Fibrillation Patients Presenting to the Emergency Department

There were no differences found between the Pre-Initiation and Post-Initiation of The Critical Pathway for Atrial Fibrillation Groups for heart rate control. Heart rate control is the initial step in the treatment management of AF prescribed in The Critical Pathway for Atrial Fibrillation. Management of AF varies depending on the severity of the manifested symptoms associated with the onset of AF, not the actual heart rate per minute. For those individuals presenting with signs of hypoperfusion, severe heart failure or unrelenting angina pain, immediate DCC was recommended by The Critical Pathway for Atrial Fibrillation. However, for those individuals presenting with a stable hemodynamic status, pharmacological rate control is indicated (Khairy & Nattel, 2002) and reflected in The Critical Pathway for Atrial Fibrillation as best practice. Physicians in the ED prescribed appropriate medications and doses based on The Critical Pathway for Atrial Fibrillation, both Pre and Post-Initiation of The Critical Pathway for Atrial Fibrillation in those individuals presenting with AF < or > 48 hours.

The ideal medications for pharmacologically controlling heart rate for those individuals with AF whom are hemodynamically stable as suggested by The Critical Pathway for Atrial Fibrillation included amiodarone, beta-blockers, calcium-channel blockers, and, inotropic drugs. Medications are ordered based on pre-existing co-morbidities, tolerance, and heart function. For those AF individuals with a preserved heart function (EF > 40%), The Critical Pathway for Atrial Fibrillation recommended the use of diltiazem, metoprolol or digoxin to lower the heart rate to $\leq$ 90 beats/minute. In those individuals, with an impaired heart function (EF < 40%), drugs of choice as

recommended by The Critical Pathway for Atrial Fibrillation included diltiazem or digoxin. In both the Pre-Initiation and Post-Initiation of The Critical Pathway for Atrial Fibrillation Groups, ideal pharmacological agents were ordered based on co-morbidities and heart function, and therefore, no differences were noted between groups, as the ED physicians were following best practice guidelines.

However, amiodarone intravenously was recommended by The Critical Pathway for Atrial Fibrillation as an agent for both the rate control and pharmacological conversion, as a loading dose followed by an oral maintenance dose for those individuals with a high risk for recurrence of AF. Although the option to prescribe amiodarone intravenously and orally for both heart rate control (as well as a conversion agent) was present in The Critical Pathway for Atrial Fibrillation, this medication was never ordered. The rationale reported by ED physicians for the refusal to order amiodarone, was based on limited published trials suggesting the limited drug's effectiveness for treating acute episodes of AF. Oral amiodarone is being prescribed with increasing frequency to treat AF, offering more effectiveness than sotalol or propafenone in preventing recurrent AF (Siddoway, 2003). Amiodarone administered intravenously, although recommended for the emergency treatment of ventricular arrhythmias (AHA, 2001), is also being recommended frequently for the treatment of acute AF, as the onset is less than 30 minutes (Siddoway, 2003).

Rhythm Control for Atrial Fibrillation Patients Presenting to the Emergency Department

Rhythm control was the second choice for treatment as outlined in The Critical Pathway for Atrial Fibrillation. This approach was similarly initiated between groups;

51.67% in the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group and 55.00% in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group. This treatment was appropriately initiated in both groups, based on The Critical Pathway for Atrial Fibrillation. However, rhythm control as a second choice was only recommended for those individuals who were hemodynamically stable in The Critical Pathway for Atrial Fibrillation. If an individual presented to the ED and was hemodynamically compromised, The Critical Pathway for Atrial Fibrillation indicated DCC for those individuals with AF < 48 hours as the first option for treatment negating heart rate control. The only difference indicated by The Critical Pathway for Atrial Fibrillation for individuals with AF > 48 hours, was the addition of an anticoagulation agent (unfractionated heparin or low molecular heparin) prior to DCC.

For those individuals who were hemodynamically stable on admission to the ED and presented with AF < 48 hours, IV amiodarone was recommended as an agent for both heart rate control and pharmacological conversion by The Critical Pathway for Atrial Fibrillation. If amiodarone was not available, agents recommended by The Critical Pathway for Atrial Fibrillation based on pre-existing co-morbidities were Class IIa antiarrhythmic agents. If heart function was preserved in individuals with AF (EF > 40%) the pathway recommended flecainide (oral), procainamide (IV), or propafenone (oral) as agents for pharmacological conversion. However, if the heart function was impaired (EF < 40%) in individuals with AF, amiodarone was recommended by The Critical Pathway for Atrial Fibrillation as a pharmacological agent. For those individuals with AF > 48 hours, pharmacological conversion was not recommended by

The Critical Pathway for Atrial Fibrillation due to the possibility of thrombus formation and risk for an embolic event.

There were no significant differences in prescribed antiarrhythmic agents between the two groups. In the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group, 21 individuals were prescribed a pharmacological agent, contrasted to 17 individuals in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group ($p = 0.56$). No difference was found in the pharmacological agent of choice in Group A and Group B, as both groups equally had IV procainamide prescribed ($p = 0.56$).

Direct current cardioversion was equally ordered between Group A ($n = 17$) and Group B ($n = 18$) ($p = 0.89$). Consideration of an antiarrhythmic agent prior to or simultaneously with DCC was recommended by The Critical Pathway for Atrial Fibrillation for those individuals with recurring AF or high risk for recurrence (long standing HTN or HF).

There were 10 individuals in Group A, who received both a pharmacological agent and DCC. Pharmacological agents were ordered as first line of treatment for rhythm control in 7 individuals (6 were given IV procainamide and 1 was given both IV procainamide and oral propafenone) without success, therefore, DCC was implemented to convert the rhythm. The other 3 individuals were ordered DCC as the first choice for rhythm conversion, which was unsuccessful and required a pharmacological agent (2 had only IV procainamide and 1 had both IV procainamide and oral propafenone) to ensure successful conversion.

In Group B, 11 individuals received both DCC and a pharmacological agent. All 11 individuals received procainamide IV as the pharmacological agent of choice, prior to DCC cardioversion. Direct current cardioversion was chosen following unsuccessful cardioversion by pharmacological means (n = 10); in the remaining individual, IV procainamide was aborted due to symptomatic hypotension.

No relationship existed between the use of The Critical Pathway for Atrial Fibrillation versus varying conventional treatment regimes and conversion from AF to SR. The drug of choice ordered by the ED physicians, as previously mentioned, was IV procainamide followed by an oral dose of propafenone, prescribed appropriately based on the patient's co-morbidities and heart function, as suggested by The Critical Pathway for Atrial Fibrillation. There were no significant differences noted as ED physicians were implementing best practice guidelines.

There was no difference between groups following the initiation of The Critical Pathway for Atrial Fibrillation, with respect to successful conversion rates ($p = 0.78$). The conversion success rate (with intervention and spontaneously) in the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group was 71.67% (n = 43) versus 68.33% (n = 41) in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group. There were 17 individuals in Group A who were unable to convert following rhythm control treatment, with a similar number in Group B (n = 18). The rates for successful cardioversion in the study are similar to those reflected in other trials (termination of AF occurs in 70 – 90% of cases, AF reoccurs in up to 50 – 75%, following initial conversion to SR), suggestive of ED physicians implementing best practice guidelines.

As previously mentioned, an option was given to ED physicians to order amiodarone both orally and IV to attempt conversion pharmacologically from AF to sinus rhythm. However, ED physicians were reluctant to order amiodarone with refusal reported to be based on limited trials demonstrating the drug's effectiveness for treating acute episodes of AF. A recent meta-analysis of randomized trials has suggested that amiodarone is as safe and effective as the class IC antiarrhythmic agents at 24 hours, offering an alternative in drug treatment for those individuals who are not acceptable candidates for rapid-acting agents (Chevalier, 2003).

Appropriate Anticoagulation for Atrial Fibrillation Patients Presenting to the Emergency Department

There were no significant differences found in appropriate anticoagulation in individuals presenting with AF, Pre versus Post-Initiation of The Critical Pathway for Atrial Fibrillation. Anticoagulation is recommended in those individuals presenting with AF > 48 hours who are hemodynamically unstable and require emergent DCC, due to a high risk for emboli formation as recommended by The Critical Pathway for Atrial Fibrillation. For those individuals presenting with AF > 48 hours who are hemodynamically stable, rate control is recommended and either a 3 week regime of coumadin prior to elective DCC, or if elective DCC is not an option, then a continual regime of anticoagulation therapy.

In the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group, appropriate anticoagulation prior to DCC was received by both individuals presenting with AF > 48 hours who were hemodynamically unstable. The first individual received IV heparin and the second individual received unfractionated heparin (enoxaparin) pre

DCC, both recommendations suggested by The Critical Pathway for Atrial Fibrillation. In the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group, three individuals presenting with AF > 48 hours who were hemodynamically stable, were treated with rate control medications and given an order for coumadin on discharge, as per The Critical Pathway for Atrial Fibrillation. There were no differences between groups, because ED physicians were implementing best practice guidelines, outlined in The Critical Pathway for Atrial Fibrillation.

Length of Stay of Atrial Fibrillation Patients Presenting to the Emergency Department

There was no relationship between the use of The Critical Pathway for Atrial Fibrillation and length of stay in the Emergency Department. The majority of individuals stay was less than 24 hours prior to discharge from care. Length of stay also included follow-up care. Follow-up care included the following variables: cardiology consults, admission to an in-patient bed in the health region, scheduling of a transesophageal echocardiogram, scheduling of an elective DCC, prescribing appropriate anticoagulation, and follow-up with a family physician.

However, there was a significant difference between groups with respect to the ordering of follow-up care for AF individuals. In the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group, 24 individuals (40.00%) compared to 36 (60.00%) individuals in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group, received follow-up care ($p = 0.04$). The most frequently ordered follow-up care component was a cardiology consult, an option recommended by The Critical Pathway for Atrial Fibrillation. There were 14 individuals (23.33%) in both Group A and Group B who received a cardiology consult. These consults were further

broken down into two groups, those individuals seen as in-hospital consults and individuals seen as out-patients post discharge from the ED.

In Group A, AF individuals not responding to regimes recommended in The Critical Pathway for Atrial Fibrillation, or who presented with AF as a result of angina, an evolving MI, or flash pulmonary edema received in-hospital cardiology consultations (n = 5). The second group consisted of individuals who required risk stratification or further investigational tests (MIBIs, TEEs, or cardiac angiograms) and were stable were referred to a cardiologist as an outpatient post-discharge (n = 9). Similar findings were found in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group, where four individuals had in-patient cardiology consultations, and 10 individuals saw a cardiologist post-discharge for either follow-up tests of risk stratification. There were no differences between groups, as ED physicians followed appropriate recommendations outlined by The Critical Pathway for Atrial Fibrillation.

Admission to an in-patient unit was another recommendation by The Critical Pathway for Atrial Fibrillation for further investigations and/or stabilization. There were no differences between groups, with five individuals in each group being admitted to either the coronary care unit, a medicine or surgical bed based on clinical findings and presentation. There were no differences between groups, because individuals, who required admission to an in-patient unit, were admitted as recommended by The Critical Pathway for Atrial Fibrillation. Therefore, the ED physicians implemented best practice for those AF individuals.

The third option for follow-up was a schedule appointment for a echocardiogram (2D or TEE) as an out-patient as recommended by The Critical

Pathway for Atrial Fibrillation. There were no significant differences noted between the two groups, as echocardiograms were ordered appropriately by either the ED physician or consulted cardiologist, as outlined in The Critical Pathway for Atrial Fibrillation. More echocardiograms were ordered by the ED physician, in the Pre-Initiation of the Critical Pathway Group (n = 9) as compared to the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group (n = 5) ($p = 0.13$). These echocardiograms were tabulated separate from those ordered by consulted cardiologists, therefore 18/60 (30.00%) individuals in the Pre-Initiation of The Critical Pathway for Atrial Fibrillation Group and 15/60 (25.00%) individuals in the Post-Initiation of The Critical Pathway for Atrial Fibrillation Group received an echocardiogram as a component of their care.

Outpatient DCC was under utilized in both Group A (n = 2) and in Group B (n = 1). Although there was no significant difference between groups ($p = 1.00$), this inappropriate utilization resulted in AF individuals (n = 15) not receiving optimal care based on The Critical Pathway for Atrial Fibrillation. In Group A, 18 individuals were unable to remain in SR post pharmacological or DCC, of these 18 individuals, 5 should have been ordered an elective DCC as recommended by The Critical Pathway for Atrial Fibrillation. All 5 individuals were discharged, 2 with cardiologist visits, and only one being treated with warfarin, although they all remained in AF.

Of the 17 individuals in Group B who were unable to convert to SR, 10 were eligible for elective DCC as outpatients as they presented with either first occurrences or intermittent AF. Only one individual (75 years of age with no identifiable risk factors for AF) presenting with chronic paroxysmal AF was treated with a 3-week

regime of coumadin prior to DCC and was sent home on 4-weeks of coumadin, appropriate practice outlined in The Critical Pathway for Atrial Fibrillation.

Although there were no significant differences noted between Group A and Group B with respect to the ordering of elective DCC as a recommended option in The Critical Pathway for Atrial Fibrillation, these results do not indicate best practice was implemented. An attempt to maintain SR in these eligible individuals could have decreased their risk for atrial remodeling, the development of atrial tachycardias, increased their maximal exercise capacity, decreased left atrial dimensions, increased left ventricular ejection fraction, and possibly improved quality of life – free of fatigue, exertional shortness of breath and/or angina (Braunwald, Zipes, & Libby, 2001; Fuster & Rydén, 2001; Gardner & Gilbert, 1996; Laupacis & Cuddy, 1996; Singh, 2000).

Although there was no difference found between groups with respect to appropriate anticoagulation for individuals presenting with AF to the ED ($p = 0.83$), this does not suggest best practice was implemented. Of the 13 individuals in the Pre-Initiation of The Critical Pathway for Atrial Fibrillation presenting with AF > 48 hours, two were sent home still in AF without a prescription for an anticoagulation medication. Since embolization occurs in 1.2% of patients who are not anticoagulated, anticoagulation of 3 weeks duration is recommended prior to DC cardioversion for AF greater than 48 hours due to adhesion of atrial thrombus to the atrial wall, since thrombus formation is thought to occur over a 2-week period. As previously, mentioned, the underutilization of anticoagulation in patients high-risk for emboli continues to present a problem (Connolly & Turpie, 1996; Lip & Li-Saw-Hee, 2000; The Heart Organization, 1999).

Emergency Department Healthcare Professional's Satisfaction with The Critical Pathway for Atrial Fibrillation

To determine if there was a relationship between ED healthcare professionals' satisfaction and outcomes of AF with the introduction of The Critical Pathway for Atrial Fibrillation, a five-item Likert questionnaire with an option to comment further, was administered to both ED Physicians and Registered Nurses (RN). The development and implementation of The Critical Pathway for Atrial Fibrillation derived from evidenced-based clinical guidelines was expected to enhance current conventional AF treatment regimes in the ED. Nine (60.00%) physicians responded, but there were no responses from the RN staff, even with multiple follow-ups.

Responses to the clarity and completeness of The Critical Pathway for Atrial Fibrillation were more favorable. With respect to the clarity of The Critical Pathway for Atrial Fibrillation, 55.6% of respondents stated the pathway was clear to follow. Those who stated the pathway was not clear to follow did not offer any suggestions on improvement of the pathway. Thoroughness of treatment also received a favorable response, with 55.6% of the respondents stating the pathway was thorough in the treatments suggested. However, the question arising from the overwhelming response (over 44 %) of ED physicians stating that The Critical Pathway for Atrial Fibrillation was not complete is, "How familiar are ED physicians with evidenced-base practice for the treatment of AF, since The Critical Pathway for Atrial Fibrillation is based on existing published guidelines?" This question is reflected in the comment from one of the respondents, "Don't need PTT or INR as baseline."

While developing The Critical Pathway for Atrial Fibrillation, three ED physicians were asked to critique and offer suggestions during the final draft of the pathway. These suggestions were implemented into the final draft of the pathway. Even with this input, only 33.33% responded favorably to the question regarding the simplicity of The Critical Pathway for Atrial Fibrillation to initiate. No rationale was offered as to why the pathway was difficult to initiate, although during the in-service phase of the study, at least two physicians suggested the pathway's length should be no longer than one page. A less than favorable response occurred in the usefulness to practice component, with 44.4% of individuals offering no opinion, and 44.4% disagreeing and 11.1% strongly disagreeing with usefulness to practice. This same unfavorable response was present in the helpfulness to practice question, with 77.8% offering no opinion, and 22.2% stating it would not be helpful to their practice. No additional comments were offered as to the rationale for these unfavorable responses.

The Critical Pathway for Atrial Fibrillation was arranged for simplicity, clarity, and completeness for implementation by the ED physician. The expectation of the initiation of the pathway in the ED, is that is would increase health care professionals' satisfaction. Further follow-up is necessary to illicit feedback from those ED physicians who worked with The Critical Pathway for Atrial Fibrillation, to ensure the pathway becomes more beneficial and valuable to ED physicians in their practice. Further constructive feedback is warranted by ED physicians familiar with the study, as well as other health care professionals whom may find The Critical Pathway for Atrial Fibrillation useful to their practice.

Limitations of the Study

One limitation of the study was the number of charts reviewed. The small numbers in each group (n = 60) may not have reflected true differences between the two groups. Comparable admission characteristics (age, gender, length of time in hours of AF prior to presentation to the ED, classification of AF into < or > 48 hours, time to see an ED physician, baseline vital signs, risk factors for the development of AF, and admission medications) may have been skewed with individuals presenting to the ED with AF in both the Pre-Initiation of The Critical Pathway for Atrial Fibrillation and the Post-Initiation of The Critical Pathway for Atrial Fibrillation Groups. A true variance may not have been reflected during the Post-Initiation of The Critical Pathway, as there were three individuals presenting twice and two individuals presenting three times during this enrollment.

Another limitation was the data collection method. The rationale for choosing a health record review was the ability to access all individuals presenting to ED with AF, anonymity of the subjects, an inexpensive method to gather data, and a hands-off approach to direct patient care from the investigator, thereby allowing ED healthcare professionals to perform in a non-intrusive manner. However, this method's limitations included a lack of direct contact between the health care professionals and the researcher, an impersonal nature, missing data, and an inability to determine rationale (if not written) for deviations in recommended care from The Critical Pathway for Atrial Fibrillation. Also the researcher cannot be confident that the ED physician followed the protocol, since the researcher assumed that placement of the guidelines on the patient's chart was indicative of use.

Implications for Current Practice

This was the first study to develop, initiate, and assess final outcomes of a critical pathway for AF derived from published clinical guidelines for the treatment of AF (The Critical Pathway for Atrial Fibrillation – Appendix A). Previously, there was no existing research that examined the application of clinical guidelines to practice and final outcomes achieved. Previous practice was based on the individual's presenting clinical symptoms and hemodynamic state, as well as physician preference.

The goals of the current study were to determine whether there was a statistically significant difference in diagnostic and treatment approach in AF individuals presenting to an ED with the initiation of The Critical Pathway for Atrial Fibrillation. There was a statistically significant difference with respect to both the number of AF occurrences ($p = 0.04$), and follow-up care ($p = 0.04$) between The Pre-Initiation of The Critical Pathway for Atrial Fibrillation and The Post-Initiation of The Critical Pathway for Atrial Fibrillation Groups, though the remaining research hypotheses were poorly supported in the study.

The final outcome of the study did not demonstrate a statistically significant difference with the initiation of The Critical Pathway for Atrial Fibrillation. There were no apparent differences in practice between groups, following initiation of The Critical Pathway for Atrial Fibrillation, suggesting the pathway based on existing guidelines was not viewed as necessary in clinical practice, and therefore, not implemented as expected by the ED health care professionals. However, the lack of differences between the two groups does not suggest best practice was initiated in the care of all individuals presenting to this ED with AF; unfavorable trends in practice continue to occur (i.e.,

inappropriate follow-up and the lack of ordering PT/INR and appropriate anticoagulation for those at high risk for thrombus formation); a common phenomenon in practice (Hunt, Baker, & Chin, 2001).

These current results support a common belief that diagnostic and treatment approaches continue to vary and optimum treatment remains ambiguous (Khairy & Nattel, 2002; Savelieva & Camm, 2000). Overall results from this study support the further application of The Critical Pathway for Atrial Fibrillation by other health care professionals at other institutions and/or health care regions. This application would determine if there are true differences due to initiation of the pathway, also allowing larger numbers of health care professionals in other EDs the opportunity to critique and offer constructive feedback of the guidelines developed for this study, thereby ensuring these guidelines are simple to initiate, clear to follow, thorough, useful to practice and helpful in providing care for AF patients.

Recommendations for the improvement of The Critical Pathway for Atrial Fibrillation include the addition and updating of research findings to reflect current evidenced-based clinical guidelines. Although anticoagulation is recommended in the high risk population, under-utilization continues to occur for fear of intracranial hemorrhages (Bungard, Ghali, Teo, McAlister, & Tsuyuki, 2000). Research and development of new agents that are as or more effective than warfarin without the adverse effects are warranted, especially in the AF population where anticoagulation is essential (i.e., ximelagatran, may be as effective as warfarin, without the adverse effects) (as cited by Fang & Singer, 2004).

Recent studies (Rate Control vs. Electrical Cardioversion for Persistent Atrial Fibrillation – RACE, and Atrial Fibrillation Follow-up Investigation of Rhythm

Management - AFFIRM), suggest rate control with long-term anticoagulation therapy is probably safer and a more effective practice versus sinus rhythm control for its own sake (AFFIRM, 2002; Kovacs, 2003; Mehta & Greenspon, 2003; Saseen, 2003; Saxonhouse & Curtis, 2003; Van Gelder, Hagens, Bosker, et al., 2002; VerNooy & Mounsey, 2004). Findings from the RACE Trial suggest that rate control is not inferior to rhythm control for persistent AF for identified endpoints (mortality, cardiovascular-related deaths, thromboembolic events, bleeding episodes, symptoms, and quality of life), and may have fewer proarrhythmia effects (Van Gelder, Hagens, Bosker, et al., 2002; Wyse, Waldo, DiMarco, et al., 2002). Another positive rationale for accepting rate control over conversion to sinus rhythm is that there is no comparable difference between rate control and rhythm control with respect to stroke, mortality, exercise tolerance, or quality of life, although quality of life in individuals with AF continues to be a concern (Hagens, Ranchor, Van Sonderen, Bosker, Kamp, Tijssen, Kingma, Harry, Crijns, & Van Gelder, 2004; Tamariz & Bass, 2004; VerNooy & Mounsey, 2004). Utilizing rhythm control versus treatment with rate control, an individual runs the risk of increase proarrhythmias, possible sinus node dysfunction and heart failure, as well as other iatrogenic risks (Wijffels & Crijns, 2004). Therefore, successful treatment of recurring AF may be to decrease symptoms rather than complete elimination of AF. Both RACE and AFFIRM suggest the optimal rate control agent may be beta-blockers, as cardiac disease and HTN are common in the AF population. Although the findings from both the RACE and AFFIRM studies suggest rate control over rhythm control as an appropriate end point, these findings may not be generalizable to the current population in this study, and therefore, may need to be incorporated into The Critical Pathway for Atrial Fibrillation with caution. However, the

findings may be relevant to certain groups of individuals, bearing in mind that younger individuals may still benefit from more aggressive treatment.

The addition of an ACE-I to a drug regime for AF may also be of benefit. The triggering of the renin-angiotensin cascade appears to play a significant role in HF related atrial remodeling. This remodeling, as well as prevent of arrhythmogenic fibrosis may be diminished with the treatment with an ACE-I (Khairy & Nattel, 2002).

Conclusion

Providing optimal care for the AF individual presenting to the ED is important and impacts clinical practice. Providing optimal care requires the health care professional to incorporate current evidenced-based clinical guidelines into their practice. The health care professional has frequent contact with individuals with recurring or chronic AF (Catherwood, Fitzpatrick, Greenberg, Holzberger, Malenka, Gerling, et al., 1999; Maglio, Ayers, Tidball, & Akhtar, 1996). With the increasing prevalence of AF individuals presenting to a health care professional, the development of a critical pathway offers a diagnostic and treatment approach derived from evidenced-based clinical guidelines to guide optimal care for the AF individual.

The Critical Pathway for Atrial Fibrillation as initiated in the GNCH ED, offered specific steps to guide healthcare professionals to ensure optimal care of an individual presenting with AF. However, in four identified components reviewed for this study (lack of ordering PT/INR, under-utilization of anticoagulation regimes, absence of administration of amiodarone, and follow-up care), optimal care was not provided for AF individuals, as evidenced by lack of initiation of The Critical Pathway

for Atrial Fibrillation. This lack of optimal care may have exposed individuals with AF unjustly to an increased risk of stroke, polypharmacy, and other complications

Individuals presenting to the ED with AF expect optimal care from health care professionals. Since best clinical practice should be reflective of evidenced based guidelines, the lack of adherence to the recommended evidence-based guidelines for the treatment of AF (American Heart Association, 2001; Canadian Cardiovascular Society Summary Statement, 1996) in this study raises ethical concerns. This study was developed with the assumption that the current guidelines (American Heart Association, 2001; Canadian Cardiovascular Society Summary Statement, 1996) were not being implemented in practice. The assumption was supported by this study, and therefore, awareness of the lack of application of best practice needs to be conveyed to all parties involved to ensure best practice is implemented. Findings from the Atrial Fibrillation Critical Pathway Questionnaire were less than favorable suggesting that physicians may not be interested in The Critical Pathway for Atrial Fibrillation to improve upon current practice, and ensure best clinical practice. This lack of interest may lie with the physicians' assumption that current practice is best practice. Since there was no change in practice, a doubt has been raised as to the acceptance and appropriateness of this pathway for use in clinical practice in the GNCH ED.

The lack of adherence or perhaps awareness of existing evidence-based guidelines does create an ideal situation for the establishment of a critical pathway to ensure that the diagnostic and treatment approach for AF is standardized without ambiguity and incongruity. The Critical Pathway for Atrial Fibrillation also offers an opportunity to educate health care professionals on "best practice", although this study

did not find any differences between groups with the initiation of The Critical Pathway for Atrial Fibrillation in the GNCH ED. The most important consequence is that it allowed for and recognized variations in individuals presenting to the GNCH ED with AF (i.e., risk factors, heart function, and admission medications) and the resultant treatment and outcome. Therefore, the initiation of The Critical Pathway for Atrial Fibrillation has the potential to guide the diagnostic and treatment approach of AF to assure care is standardized, reduces complications and morbidity, and introduces potential cost-effective measures – ensuring initiation of best clinical practice.

References

Albers, G. W., Dalen, J. E., Laupacis, A., Manning, W. J., Petersen, P., & Singer, D. E. (2001). Antithrombotic therapy in atrial fibrillation. *Chest*, 119, 194S - 206S.

American Heart Association in collaboration with the International Liaison Committee on Resuscitation (2001). Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care. Part 6: advanced cardiovascular life support. *Circulation*, 102(Suppl 1): 186 - 171. Retrieved from the World Wide Web May 17, 2002: www.circ.ahajournals.org/cgi/content/full/102/suppl_1/1-112#T2

Arnsdorf, M. F., & Lip, G. Y. H. (2000). Anticoagulation to prevent embolization in chronic atrial fibrillation: Clinical trials. *Copyright 2000 Up-To-Date®, Inc.*, April 28, 2000.

Arnsdorf, M. F., & Lip, G. Y. H. (2000). Anticoagulation to prevent embolization in chronic atrial fibrillation: Recommendations. *Copyright 2000 Up-To-Date®, Inc.*, April 18, 2000.

Arnsdorf, M. F. (2000). Causes of atrial fibrillation. *Copyright 2000 Up-To-Date®, Inc.*, May 23, 2000.

Asher, C. R., Miller, D. P., Grimm, R. A., Cosgrove, D. M., & Chung, M. K. (1998). Analysis of risk factors for development of atrial fibrillation early after cardiac valvular surgery. *The American Journal of Cardiology*, 82: 892 – 895.

Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) Investigators (2002). A comparison of rate control and rhythm control in patients with atrial fibrillation. *The New England Journal of Medicine*, 347: 1825 – 1833.

Awtry, E. H., & Loscalzo (2001). Cardiac arrhythmias. In Andreoli, T. E., Carpenter, C. C. J., Griggs, R. C., & Loscalzo, J. (Eds.), *Cecil Essentials of Medicine*, 5th ed., p 100 – 126. Philadelphia, PA: W. B. Saunders Company.

Bharucha, D. B., Kowy, P. R. (2000). A Symposium: AFIB – Advances for the New Millennium: Management and prevention of atrial fibrillation after cardiovascular surgery. *The American Journal of Cardiology*, 85 (10A): 20D – 24D.

Bracken, J. (1997). Reducing door-to-needle time: Treatment delay versus presentation delay. *Clinical Cardiology*, 20(Suppl 3), 21 - 25.

Botteron, G. W. & Smith, J. M. (1998). Cardiac arrhythmias. In Carey, C. F., Lee, H. H., & Woeltje, K. F. (Eds.), *The Washington Manual of Medical Therapeutics* (29th ed.), 134 – 135. Philadelphia: Lippincott, Williams & Wilkins.

Braunwald, E., Zipes, D. P., & Libby, P. (2001). *Heart Disease: A Textbook of Cardiovascular Medicine,* (6th ed.). Philadelphia: W. B. Saunders Company.

Bungard, T. J., Ghali, W. A., Teo, K. K., McAlister, F. A., & Tsuyuki, R. T. (2000). Why do patients with atrial fibrillation not receive warfarin? *Archives of Internal Medicine,* 160: 41 – 46.

Cairns, J. A., Theroux, P., Lewis, H. D., Ezekowitz, M., Meade, T. W., & Sutton, G. C. (1998). Antithrombotic agents in coronary artery disease. *Chest,* 114(5): Suppl 611S – 633S.

Camm, A. J. (2001). Atrial fibrillation: Is there a role for low-molecular-weight heparin? *Clinical Cardiology*, 24(Suppl 3): 15 - 19.

Canadian Cardiovascular Society (1996). Summary statement. *Canadian Journal of Cardiology*, 12(Suppl A), 51A - 57A.

Cannom, D. S. (2000). A Symposium: AFIB – Advances for the New Millennium: Atrial fibrillation: Nonpharmacologic Approaches. *The American Journal of Cardiology*, 85(10A): 25D – 35D.

Cappuci, Al, Lenzi, T., Boriani, G., et al. (1992). Effectiveness of loading oral flecainide for converting recent-onset atrial fibrillation to sinus rhythm in patients without organic heart disease or with only systemic hypertension. *American Journal of Cardiology*, 70: 69 - 72.

Caro, J. J., Flegel, K. M., Oreguela, M., Kelley, H. E., Speckman, J. L., & Migliaccio-Walle, K. (1999). Anticoagulant prophylaxis against stroke in atrial fibrillation: Effectiveness in actual practice. *Canadian Medical Association Journal*, 161(5), 493 - 497.

Catherwood, E., Fitzpatrick, W. D., Greenberg, M. L., Holzberger, P. T., Malenka, D. J., Gerling, B. R., et al. (1999). Cost-effectiveness of cardioversion and antiarrhythmic therapy in nonvalvular atrial fibrillation. *Annals of Internal Medicine*, 130: 625 – 636.

Chakko, S., & Myerburg, R. J. (1999). Arrhythmias and conduction disturbances. In, Alexander, R. W., Schlant, R. C., Fuster, V., O'Rourke, R. A., Roberts, R., & Sonnenblick, E. H. (Eds.), *Hurst's The Heart: Companion Handbook*, 9th ed., p 17 - 53. New York, NY: McGraw-Hill Health Professions Division.

Cheng, J. (1999). Arrhythmia. In Singleton, J. K., Sandowski, S. A., Green-Hernandez, C., Horvath, T. V., DiGregorio, R. V., & Holzemer, S. P. (Eds.), *Primary Care*, pp. 61 – 76. Philadelphia: Lippincott.

Chevalier, P., et al., (2003). Amiodarone versus placebo and class 1c drugs for cardioversion of recent-onset atrial fibrillation: A meta-analysis. *Journal of the American College of Cardiology,* 41: 255 – 262.

City of Edmonton (November, 2001). Edmonton population and employment forecast allocation study 2000–2025: Summary report. Retrieved from the World Wide Web May 9, 2002: www.gov.edmonton.ab.ca/planning-policy_services_branch/economic_demographic_information/pdf_files/Allocation%2oStudy_%20Summary%20Report.pdf

Cohen, N., Almoznino-Sarafin, D., Alon, I., Gorelik, O., Koopfer, M. & Chachashvily, S., et al. (2000). Warfarin for stroke prevention still underused in atrial fibrillation patterns of omission. *Stroke*, 3(1), 1217 – 1222.

Connolly, S. J. & Turpie, A. G. G. (1996). Antithrombotic therapy in atrial fibrillation. *Canadian Journal of Cardiology*, 12(Suppl A), 17A – 20A.

Cuddy, T. E. & Connolly, S. J. (1996). Atrial fibrillation and atrial flutter. *Canadian Journal of Cardiology,* 12(Suppl A), 9A – 11A.

Cotter, G., Blatt, A., Kaluski, K., et al., (1999). Conversion of recent onset paroxysmal atrial fibrillation to normal sinus rhythm: The effect of no treatment and high dose amiodarone. *European Heart Journal*, 20: 1833 - 1842.

Crystal, E. & Connolly, S. J. (2004). Atrial fibrillation: Guiding lessons from epidemiology. *Cardiology Clinics*, 22(1), 1 – 8.

Cummins, R. O. & Hazinski, M. F. (2000). Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care: International consensus on science. *Currents*, 11(3), 1 - 28. Retrieved from the World Wide Web March 13, 2002: www.currentsonline.com/adobe/fall2000.pdf

Donovan, K. D., Power, B. M., Hockings, B. E. F., Dobb, G. J., & Lee, K. Y. (1995). Intravenous flecainide versus amiodarone for recent-onset atrial fibrillation. *American Journal of Cardiology*, 75: 693 - 693.

Ellis, M. F. (1998). Atrial fibrillation following cardiac surgery. *Dimensions of Critical Care Nursing*, 17(5): 226 – 242.

Everett, T. H., & Olgin, J. E. (2004). Basic mechanisms of atrial fibrillation. *Cardiology Clinics*, 22 (1), 9 – 20.

Ezekowitz, M. D., & Levine, J. A. (1999). Preventing stroke in patients with atrial fibrillation. *JAMA*, 281(19): 1830 – 1835.

Fang, M. C., & Singer, D. E. (2004). Anticoagulation for atrial fibrillation. *Cardiology Clinics*, 22(1), 47 – 62.

Fuster, V., & Rydén, L. E., et al. (2001). ACC/AHA/ESC Guidelines for the management of patients with atrial fibrillation. *Journal of the American College of Cardiology*, 38(4) 1 – 70.

Galve, E., Rius, T., Ballester, R., et al. (1996). Intravenous amiodarone in treatment of recent-onset atrial fibrillation: Results of a randomized, controlled study. *Journal of the American College of Cardiology*, 27: 1079 - 1082.

Gardner, M. J. & Gilbert,l M. (1996). Heart rate control in patients with atrial fibrillation. *Canadian Journal of Cardiology*, 12(Suppl A), 21A – 23A.

Gillis, A. M., Klein, G. J. & MacDonald, R. G. (1996). Investigation of the patient with atrial fibrillation. *Canadian Journal of Cardiology*, 12(Suppl A), 12A – 13A.

Government of Alberta (2000). Chronic disease and injury. In, *Health Trends in Alberta 2000 – A Working Document.* Retrieved from the World Wide Web March 1, 2002 from: www.health.gov.ab.ca/public/document/Heath_Trends/index.html

Gow, R. M. (1996). CCS Consensus Conference on Atrial Fibrillation: Atrial fibrillation and flutter in children and young adults with congenital heart disease. *Canadian Journal of Cardiology*, 12 (Suppl A): 45A – 48A.

Hagens, V. E., Ranchor, A. V., Van Sonderen, C. V., Bosker, H. A., Kamp, O., Tijssen, J. G. P., Kingma, J. H., Crijns, H. G. M., & Van Gelder, I. C. (2004). Effect of rate or rhythm control on quality of life in persistent atrial fibrillation. *Journal of the American College of Cardiology*, 43(2) 241 – 247.

Haissaguerre, J., Jais, P., Shah, D. C., Takashashi, A., Hocini, M., Quiniou, G., et al. (1998). Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *The New England Journal of Medicine*, 339: 659 – 666.

Harenberg, J., Weuster, B., Pfitzer, M., Dempfle, C. E., Stehle, G., Kubler, W., & Schlierf, G. (1993). Prophylaxis of embolic events in patients with atrial fibrillation using low molecular weight heparin. *Seminars in Thrombosis & Hemostasis*, 19(Suppl 1), 116 - 121.

Hauser, S. L.(2000). Therapeutic guidelines for stroke prevention in nonvalvular atrial fibrillation. *Copyright 2000 Up-To-Date®, Inc.*, February 10, 2000.

Heart and Stroke Foundation of Canada (1999). The Changing Face of Heart Disease and Stroke in Canada 2000.

Heartwire (1999). Warfarin use is rising among atrial fibrillation patients, but probably not enough. Retrieved from the World Wide Web, February 19, 2001: http://www.theheart.org/documents/docs7000/7074/

Hebbar, A. K., & Hueston, W., J. (2002). Management of common arrhythmias: Part 1. Supraventricular arrhythmias. *American Family Physician*, 65(12), 2479 – 2486.

Hilleman, D. E., & Spinler, S. A. (2002). Conversion of recent onset of atrial fibrillation with intravenous amiodarone: A meta analysis of randomized controlled trials. *Pharmacotherapy*, 22(1), 66 - 74.

Hunter, S. A., Baker, D. W., Chin, M. H., et al., (2001). ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation: Executive summary. A report of the American College of Cardiology/American Heart Association task force on practice guidelines and policy conferences (committee to develop guidelines for the management of patients with atrial fibrillation): Developed in collaboration with the North American Society of Pacing and Electrophysiology. *Journal of the American College of Cardiology*, 38(4): 1231 – 1266.

Hylek, E. M., & Borowsky, L. H., et al. (1999). Warfarin use among ambulatory patients with nonvalvular atrial fibrillation: The AnTicoagulation and Risk Factors in Atrial Fibrillation (ATRIA) study. *Annals Internal Medicine*, 131: 927 – 934.

Kerr, C. R., Boone, J., Connolly, S. J., Dorian, P., Green, M., Klein, G., et al. (1998). The Canadian registry of atrial fibrillation: A noninterventional follow-up of patients after the first diagnosis of atrial fibrillation. *American Journal of Cardiology*, 82, 82N, 1 – 5.

Kerr, C. R. & Klein, G. J. (1996). Atrial fibrillation – Future directions. *Canadian Journal of Cardiology,* 12(Suppl A), 58A – 61A.

Kerr, C. R. & Pym, J. (1996). Pacing and atrial fibrillation. *Canadian Journal of Cardiology,* 12(Suppl A), 36A – 39A.

Khairy, P., & Nattel, S. (2002). New insights into the mechanisms and management of atrial fibrillation. *Canadian Medical Association Journal*, 167(9), 1012 – 1020.

Khan, G. M. (1997). *On Call Cardiology*, pp. 236-238. Philadelphia: W. B. Saunders Company.

King, D. E., Dickerson, L. M., & Sack, J. L. (2002). Acute management of atrial fibrillation: Part II. Prevention of thromboembolic complications. *American Family Physician*, 66(2), 2611 – 264.

Klein, G. J. & Kerr, C. R. (1996). Nonpharmacological therapy of atrial fibrillation. *Canadian Journal of Cardiology,* 12(Suppl A), 49A – 50A.

Kovacs, K. A. (2003). Long-term management of atrial fibrillation: Rhythm or rate control? *Canadian Medical Association Journal*, 168(5), 591 – 592.

Kumagai, K., Khrestian, C., & Waldo, A. L. (1997). Simultaneous multisite mapping studies during induced atrial fibrillation in the sterile pericarditis model; insights into the mechanism of its maintenance. *Circulation*, 95, 511 - 521.

Kumar, A. (1996). Intravenous amiodarone for therapy of atrial fibrillation and flutter in critically ill patients with severely depressed left ventricular function. *Southern Medical Journal*, 89(8), 779 - 785.

Lacy, C. (2000). Warfarin: Drug Information. *Copyright 2000 Up-To-Date®, Inc.*, August 8, 2000.

Laupacis, A. & Cuddy, T. E. (1996). Prognosis of individuals with atrial fibrillation. *Canadian Journal of Cardiology*, 12(Suppl A), 14A – 16A.

Lin, H. J., Wolf, P.A., Kelly-Hayes, M., Beiser, A. S., Kase, C. S., Benjamin, E. J., & D'Agostine, R. B. (1996). Stroke severity in atrial fibrillation. The Framingham Study. *Stroke*, 27(10), 1760 – 1764.

Lip, G. Y. H., & Li-Saw-Hee, F. L. (2000). Anticoagulation in the elderly. *Copyright 2000 Up-To-Date®, Inc.*, May 1, 2000.

Maglio, C., Ayers, G., Tidball, E. W., & Akhtar, M. (1996). Health care utilization and cost of care in patients with symptomatic atrial fibrillation. *Circulation*, 94(Suppl) 1 – 169.

Matthew, J. P., Parks, R., Savine, J. S., Friedman, A. S., Koch, C., Mangano, D. T., & Browner, W. S. (1996). Atrial fibrillation following coronary artery bypass graft surgery. *JAMA, 276*(4), 300 – 306.

Mehta, N. N., & Greenspon, A. J. (2003). Atrial fibrillation: Rhythm versus rate control. *Geriatrics,* 58(4), 39 – 44.

Mittal, S., Ayati, S., & Stein, K. M., et al. (2000). Transthoracic cardioversion of atrial fibrillation: Comparison of rectilinear biphasic versus damped sine wave monophasic shocks. *Circulation*, 101, 1282 - 1287.

Moore, S. L., & Wilkoff, B. L. (1991). Rhythm disturbances after cardiac surgery. *Seminars in Thoracic and Cardiovascular Surgery*, 3(1): 24 – 28.

Murray, R. D., Deitcher, S. R., Shah, A., Jasper, S. E., Bashir, M., Grimm, R. A., & Klein, A. L. (2001). Potential clinical efficacy and cost benefit of a transesophageal echocardiography-guided low-molecular-weigh heparin (enoxaparin) approach to antithrombotic therapy in patients undergoing immediate cardioversion from atrial fibrillation. *Journal of the American Society of Echocardiography*, 14(3), 200 - 208.

Nattel, S. (2002). New ideas about atrial fibrillation 50 years on. *Nature*, 415, 219 – 226.

Nattel, S., Talajie, M., Quantz, M., et al. (1987). Frequency-dependent effects of amiodarone on atrio-ventricular nodal function and slow-channel action potentials: Evidence for calcium channel blocking activity. *Circulation*, 76: 442 - 449.

Newman, D., Gillis, A., Gilbert, M. &Dorian, P. (1996). Chronic drug therapy to prevent recurrence of atrial fibrillation. *Canadian Journal of Cardiology*, 12(Suppl A), 24A – 28A.

Page, P. L., & Pym, J. (1996). CCS Consensus Conference on Atrial Fibrillation: Atrial fibrillation following cardiac surgery. *Canadian Journal of Cardiology*, 12 (Suppl A): 40A – 44A.

Paquette, M., Roy, D., Talajic, M., Newman, D., Couturier, A., Yang, C., et al. (2000). Role of gender and personality on quality-of-life impairment in intermittent atrial fibrillation. *American Journal of Cardiology*, 86, 764 - 768.

Phillips, S. (1998). Canadian Cardiovascular Society 1998 Consensus Conference on the prevention of cardiovascular diseases: The Role of the cardiovascular specialist. Retrieved from the World Wide Web February 21, 2001: http://www.ccs.ca/society/conferences/archives/1998/1998coneng-23.cfm

Prystowsky, E. N. (2000). Management of atrial fibrillation: Therapeutic options and clinical decisions. *The American Journal of Cardiology*, 85 (10A), 3D – 11D.

Prystowsky, E. N., & Klein, G. (1994). *Cardiac arrhythmias: An integrated approach for the clinician.* New York, NY: McGraw-Hill, Inc.

Raskob, G. E. (1995). Low molecular weight heparin, heparin, and warfarin. *Current Opinion in Hematology*, 2(5), 372-379.

Reiffel, J. A. (2000). A Symposium: AFIB – Advances for the New Millennium: Drug choices in the treatment of atrial fibrillation. *The American Journal of Cardiology*, 85(10A): 12D – 19D.

Reiffel, J. A. (2000). A Symposium: AFIB – Advances for the New Millennium: Formulation substitution and other pharmacokinetic variability: Underappreciated variables affecting antiarrhythmic efficacy and safety in clinical practice. *The American Journal of Cardiology*, 85(10A): 46D – 52D.

Robinson, A., & Thomson, R. G. (2000). The potential use of decision analysis to support shared decision making in the face of uncertainty: The example of atrial fibrillation and warfarin anticoagulation. *Quality in Health Care*, 9(4), 238 - 244.

Roy, D., Talajic, M., Dorian, P., Connolly, S., Eisenbery, M. J., Green, M., et al. (2000). Amiodarone to prevent recurrence of atrial fibrillation. Canadian trial of atrial fibrillation investigators. *New England Journal of Medicine*, 342: 913 - 920.

Saxonhouse, S. J., & Curtis, A. B. (2003). Risks and benefits of rate control versus maintenance of sinus rhythm. *The American Journal of Cardiology*, 91(6A): 27D – 32D.

Saseen, J. J. (2003). Is rate control better than rhythm control for atrial fibrillation in older high-risk patients? *The Journal of Family Practice*, 52(3) 192 – 194.

Savelieva, I. & Camm, A. J. (2000). Clinical relevance of silent atrial fibrillation: Prevalence, prognosis, quality of life, and management. *Journal of Interventional Cardiac Electrophysiology*, 4(2), 369 - 382.

Shevlin, J. D., Summers-Bean, C., Thomas, D., Whitney, C. G., Todd, D., & Ray, S. M. (2002). A systematic approach for increasing pneumococcal vaccination rates at an inner-city public hospital. *American Journal of Preventive Medicine*, 22(2), 92 - 97.

Shulman, R. I. (2000). Assessment of low-molecular-weight heparin trials in cardiology. *Pharmacology & Therapeutics*, 87(1), 1 - 9.

Singh, B. N. (2000). Management of atrial fibrillation. Retrieved from the World Wide Web February 19, 2001: http://cardiology.medscape.com/Medscape/CNO/2000/ACC/Story.cfm?story_id=1076.

Talajic, M., MacDonald, R. G. & Nattel, S. (1996). Restoration of sinus rhythm in patients with atrial fibrillation. *Canadian Journal of Cardiology*, 12(Suppl A), 29A – 35A.

Tamariz, L. J., & Bass, E. B. (2004). Pharmacological rate control of atrial fibrillation. *Cardiology Clinics*, 22(1), 35 – 45.

Taylor, F. C., Cohen, H., & Ebrahim, S. (2001). Systematic review of long term anticoagulation or antiplatelet treatment in patients with non-rheumatic atrial fibrillation. *British Medical Journal*, 332: 321 – 326.

Tskiouris, J. P., & Cox, C. D. (2001). A review of Class III antiarrhythmic agents for atrial fibrillation: Maintenance of normal sinus rhythm. *Pharmacotherapy*, 21(2), 1514 - 1529.

Van Gelder, I. C., Hagens, V. E., Bosker, H. A., et al. (2002). A comparison of rate control and rhythm control in patients with recurrent persistent atrial fibrillation. *The New England Journal of Medicine*, 347: 1834 – 1340.

Van Gelder, I. C., Tuinenburg, A. E., Schoonderwoerd, B. S., Tieleman, R. G., & Harry, J. G. M. (1999). Pharmacologic versus direct-current electrical cardioversion of atrial flutter and fibrillation. *The American Journal of Cardiology*, 84(9A): 147R – 151R.

VerNooy, R. A., & Mounsey, J. P. (2004). Antiarrhythmic drug therapy of atrial fibrillation. *Cardiology Clinics*, 22(1), 21 – 34.

Wentworth, D. A., Atkinson, R. P. (1996). Implementation of an acute stroke program decreases hospitalization costs and length of stay. *Stroke*, 27(6), 1040 - 1043.

Wijffels, M. C. E. F., & Crijns, H. J. G. M. (2004). Rate versus rhythm control in atrial fibrillation. *Cardiology Clinics*, 22 (1), 63 – 69.

Wolf, P. A., Abbott, R. D., & Kannel, W. B. (1991). Atrial fibrillation as an independent risk factor for stroke: The Framingham Study. *Stroke*, 22(8), 983 – 988.

Woolf, S. H., & George, J. N. (2000). Evidence-based medicine. Interpreting studies and setting policy. *Hematology – Oncology Clinics of North America*, 14(4), 761 - 784.

Wyse, D. G., Waldo, A. L., DiMarco, J. P., et al. (2002). A comparison of rate control and rhythm control in patients with atrial fibrillation. *The New England Journal of Medicine,* 347: 1825 – 1833.

Yusuf, S., Sleight, P., Pogue, J., Davies, R., & Dagenais, G. (2000). Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. The Heart outcomes prevention evaluation study investigators. *New England Journal of Medicine*, 342: 145 – 153.

Zimetbaum, P. J., & Josephson, M. E. (1999). The evolving role of ambulatory arrhythmia monitoring in general practice. *Annals of Internal Medicine*, 130(10): 848 – 856.

Zipes, D. P. (2003). Mechanisms of clinical arrhythmias. *PACE*, 26, 1778 – 1792.

Appendix A

An ER/ICU/CCU Critical Pathway for Symptomatic Atrial Fibrillation

AF <48 Hours

Preserved/Impaired Heart Function (determined by clinical findings, radiographic tests, and/or previous cardiac catherization, MUGA or TEE results)

Severely Symptomatic

Severely symptomatic refers to a **rapid ventricular response** usually greater than 150 beats per minute associated with **severe hypotension, acute pulmonary distress** or **unstable angina**

Preliminary workup prior to DC Cardioversion

_____1. 12 lead ECG
_____2. Continuous telemetry monitoring
_____3. VS including O_2 @ ___L by np prn; titrate to saturation > 90%
_____4. Initiate 1 IV site (0.9% NS @ _____cc/hr)

DC Cardioversion

_____1. Obtain consent form
_____2. Conscious sedation (titrate if using agents in combination)
  a. ____ Midazolam/Versed IV: 0.07 mg/kg
  b. ____ Fentanyl IV: 1-3 mcg/kg
  c. ____ Propofol IV: 5mcg/kg/min may increase by 5-10 mcg/kg/min q5-10 minutes until desired level of sedation is reached
_____3. Consider antiarrhythmic prior/simultaneously to DC cardioversion in individuals with recurring AF or high risk for recurrence (long standing HTN or CHF) and who are hemodynamically stable.
  a. ____ Amiodarone IV: Loading dose 5 mg/kg over 15 minutes for a maximum of 300 mg
  b. ____Amiodarone conversion dose: 3mg/min x 3 hours. Discontinue infusion if conversion to SR successful or patient becomes hemodynamically unstable)
_____4. Anterior-Posterior electrode configuration preferred
_____5. Biphasic Synchronized DC shock preferred (with assessment of cardiac rhythm after each shock)
  a. ____ Beginning at 50 joules
  b. ____ Increasing incrementally from 100 and 200 joules until normal sinus rhythm is reached

Diagnostics if warranted

_____1. Blood work:
  _____ STAT: CBC differential, sodium, potassium, chloride, CO_2, creatinine, blood urea, magnesium, and calcium
  _____ STAT: PT (INR)/PTT
  _____ Consider CK, CKMB, and Troponin if suspecting ischemia
  _____ Consider TSH
  _____ Consider toxicology screen if suspecting substance abuse
_____2. Chest x-ray

Oral anticoagulation only if at high risk for embolization (long-standing history of CHF, HTN)

_____ 1. Minimum of 3 weeks warfarin therapy pre outpatient DC cardioversion if necessary
  a. dose _____mg
  b. day to be seen by GP for monitoring of INR levels _____
_____ 2. Minimum of 4 weeks warfarin therapy post DC cardioversion
  a. dose _____mg
  b. day to be seen by GP for monitoring of INR levels _____

Follow-up Care

_____1. Consult Cardiology
_____2. Admit for further investigations and/or stabilization
_____3. Book appointment for outpatient TEE/2D echo
  DATE: _____
_____4. Book appointment for outpatient DC Cardioversion if necessary
  DATE: _____

_____5. Discharge ensuring follow-up for monitoring of INR levels if at high risk for embolization (long standing history of CHF, HTN)

Unsuccessful DC Cardioversion

_____1. **IV AMIODARONE AVAILABLE (hemodynamically stable)**

a. **Rate Control** (preferably < 90 beats/minute)

b. **Pharmacological Conversion** for all patients **(Heart Function Preserved and Heart Function Impaired)**

i. ____ Amiodarone IV: Loading dose 5 mg/kg over 15 minutes for a maximum of 300 mg

ii.____Amiodarone conversion dose: 3mg/min x 3 hours. Discontinue infusion if conversion to SR successful or patient becomes hemodynamically unstable)

iii. Oral Maintenance dose: in those individuals with high risk for recurrence of AF (numerous episodes of AF, HTN, and CHF)

____ 400 mg BID x 14 days

____ 200 mg/day

____2. **IV AMIODARONE NOT AVAILABLE**

Rate Control (preferably < 90 beats/minute)

a. **Heart Function Preserved (EF > 40%)** – Use only one of the following:

i. Initial (IV) dose

_____ Diltiazem (0.25 mg/kg) IV

_____ Metoprolol (2.5 – 5.0 mg; up to 3 doses) IV

_____ Digoxin (0.5 mg; followed by 0.25 mg q6h x 2 prn) IV

_____ Other medications used than those listed above:

ii. Maintenance (Oral) dose:

_____ Diltiazem 120-360 mg daily in divided doses

_____ Metoprolol 25-100mg BID

_____ Other medications:

b. **Heart Function Impaired (< 40%)** – Use only one of the following:

i. Initial (IV) dose

_____ Digoxin (0.5 mg; followed by 0.25 mg q6h x 2 prn) IV

_____ Diltiazem (0.25 mg/kg) IV

_____ Other medications used than those listed above:

ii. Maintenance (Oral) dose:

_____ Amiodarone 800mg daily for one week; 600 mg daily for one week; 400 mg daily for 4-6 weeks; usual maintenance dose 200 mg daily

_____ Digoxin 0.125-0.375 mg daily

_____ Diltiazem 120-360 mg daily in divided doses

_____ Other medications:

Consider oral anticoagulation if at risk for embolization
(long-standing history of CHF, HTN)

_____ 1. Minimum of 3 weeks warfarin therapy pre outpatient DC cardioversion if necessary

a. dose _____mg

b. day to be seen by GP for monitoring of INR levels _____

_____ 2. Minimum of 4 weeks warfarin therapy post DC cardioversion

a. dose _____mg

b. day to be seen by GP for monitoring of INR levels _____

Follow-up Care

_____1. Consult Cardiology

_____2. Admit for further investigations and/or stabilization

_____3. Book appointment for outpatient TEE/2D echo

DATE: _____

_____4. Book appointment for outpatient DC Cardioversion if necessary

DATE: _____

_____5. Discharge ensuring follow-up for monitoring of INR levels if at high risk for embolization **(long standing history of CHF, HTN)**

Stable

Preliminary workup

_____1. 12 lead ECG
_____2. Continuous telemetry monitoring
_____3. VS including O_2 @ ___L by np prn; titrate to saturation > 90%
_____4. Initiate 1 IV site (0.9% NS @ _____cc/hr)
_____5. Blood work:
- _____ STAT: CBC differential, sodium, potassium, chloride, CO_2, creatinine, blood urea, magnesium, and calcium
- _____ STAT: PT (INR)/PTT
- _____ Consider CK, CKMB, and Troponin if suspecting ischemia
- _____ Consider TSH
- _____ Consider toxicology screen if suspecting substance abuse

_____6. Chest x-ray
_____7. NPO in preparation for DC Cardioversion if condition warrants
_____8. Weight _____kg; Height _____cm
_____9. Consider rate control and anticoagulation in those individuals presenting with onset of AF<48 hours prior to cardioversion with a potential risk for embolization (i.e. long standing history of CHF, HTN)

IV AMIODARONE AVAILABLE

_____1. **Rate Control** (preferably < 90 beats/minute)
_____2. **Pharmacological Conversion** for all patients **(Heart Function Preserved and Heart Function Impaired)**
- a. ____ Amiodarone IV: Loading dose 5 mg/kg over 15 minutes for a maximum of 300 mg
- b. ____Amiodarone conversion dose: 3mg/min x 3 hours. Discontinue infusion if conversion to SR successful or patient becomes hemodynamically unstable)
- c. Oral Maintenance dose: in those individuals with high risk for recurrence of AF
  - ____ 400 mg BID x 14 days
  - ____ 200 mg/day

IV AMIODARONE NOT AVAILABLE

_____1. **Rate Control** (preferably < 90 beats/minute)
- a. **Heart Function Preserved (EF > 40%)** – Use only one of the following:
  - i. Initial (IV) dose
    - _____ Diltiazem (0.25 mg/kg) IV
    - _____ Metoprolol (2.5 – 5.0 mg; up to 3 doses) IV
    - _____ Digoxin (0.5 mg; followed by 0.25 mg q6h x 2 prn) IV
    - _____ Other medications used than those listed above:

  - ii. Maintenance (Oral) dose:
    - _____ Diltiazem 120-360 mg daily in divided doses
    - _____ Metoprolol 25-100mg BID
    - _____ Other medications:

- b. **Heart Function Impaired (< 40%)** – Use only one of the following:
  - i. Initial (IV) dose
    - _____ Digoxin (0.5 mg; followed by 0.25 mg q6h x 2 prn) IV
    - _____ Diltiazem (0.25 mg/kg) IV
    - _____ Other medications used than those listed above:

  - ii. Maintenance (Oral) dose:
    - _____ Amiodarone 800mg daily for one week; 600 mg daily for one week; 400 mg daily for 4-6 weeks; usual maintenance dose 200 mg daily
    - _____ Digoxin 0.125-0.375 mg daily
    - _____ Diltiazem 120-360 mg daily in divided doses
    - _____ Other medications:

____ 2. **Convert Rhythm - Pharmacological conversion**
- a. **Heart Function Preserved (>40%)** – Use one of the following:
  - i. Initial dose
    - _____ Flecainide oral (100mg q12 h)

_____ Procainamide IV(100 mg q5min @ rate of 25-50 mg/min until arrhythmia is controlled or 1 g given, then 2-6 mg/min)
_____ Propafenone oral(450 – 600 mg)
_____ Other medications:

b. **Heart Function Impaired (<40%)**
i. Please list other medications used than recommended Amiodarone:

Oral anticoagulation only if at high risk for embolization (long-standing history of CHF, HTN)

_____ 1. Minimum of 3 weeks warfarin therapy pre outpatient DC cardioversion if necessary
a. dose _____mg
b. day to be seen by GP for monitoring of INR levels _____
_____ 2. Minimum of 4 weeks warfarin therapy post DC cardioversion
a. dose _____mg
b. day to be seen by GP for monitoring of INR levels _____

Follow-up Care

_____1. Consult Cardiology
_____2. Admit for further investigations and/or stabilization
_____3. Book appointment for outpatient TEE/2D echo
DATE: _____
_____4. Book appointment for outpatient DC Cardioversion if necessary
DATE: _____
_____5. Discharge ensuring follow-up for monitoring of INR levels if at high risk for embolization (long standing history of CHF, HTN)

An ER/ICU/CCU Critical Pathway for Symptomatic Atrial Fibrillation

AF >48 Hours

Preserved/Impaired Heart Function (determined by clinical findings, radiographic tests, and/or previous cardiac catherization, MUGA or TEE results)

Severely Symptomatic

> **Severely symptomatic** refers to a **rapid ventricular response** usually greater than 240 beats per minute **and/or** associated with **severe hypotension, acute pulmonary distress** or **unstable angina**

Preliminary workup prior to DC Cardioversion

_____1. 12 lead ECG
_____2. Continuous telemetry monitoring
_____3. VS including O_2 @ ___L by np prn; titrate to saturation > 90%
_____4. Initiate 1 IV site (0.9% NS @ _____cc/hr)

DC Cardioversion

_____1. Obtain consent form
_____2. Anticoagulation Intravenous (unless contraindicated)
- a. Unfractionated Heparin
  - ____ Bolus as per Cardiology nomogram (#3830-03332)
  - ____ IV heparin 25, 000 units in 250cc of D5W rate as per cardiology nomogram (#3830-03332) and titrate according to this nomogram
- b. Low Molecular Weight Heparin
  - ____ Dalteparin (Fragmin®) _____units (200 units/kg) sc initial dose (maximum dose: 18,000 units in divided doses)
  - ____ Enoxaparin (Lovenox®) _____mg (1.5mg/kg) sc initial dose (maximum daily dose: 100 mg in divided doses)

_____3. Consider antiarrhythmic prior/simultaneously to DC cardioversion
- a. ____ Amiodarone IV: Loading dose 5 mg/kg over 15 minutes for a maximum of 300 mg
- b. ____ Amiodarone conversion dose: 3mg/min x 3 hours. Discontinue infusion if conversion to SR successful or patient becomes hemodynamically unstable)

_____4. Conscious sedation (titrate if using agents in combination)
- a. ____ Midazolam/Versed: 0.07 mg/kg IV
- b. ____ Fentanyl: 1-3 mcg/kg IV
- c. ____ Propofol: 5mcg/kg/min may increase by 5-10 mcg/kg/min q5-10 minutes until desired level of sedation is reached IV

_____5. Anterior-Posterior electrode configuration preferred
_____6. Biphasic Synchronized DC shock preferred (with assessment of cardiac rhythm after each shock)
- a. ____ Beginning at 50 joules
- b. ____ Increasing incrementally from 100 and 200 joules until normal sinus rhythm is reached

If successful, further diagnostics if warranted

_____1. Blood work:
- _____ STAT: CBC differential, sodium, potassium, chloride, CO_2, creatinine, blood urea, magnesium, and calcium
- _____ STAT: PT (INR)/PTT
- _____ Consider CK, CKMB, and Troponin if suspecting ischemia
- _____ Consider TSH
- _____ Consider toxicology screen if suspecting substance abuse

_____2. Chest x-ray

Oral Anticoagulation

_____ 1. Minimum of 3 weeks warfarin therapy pre outpatient DC cardioversion if necessary
- a. dose _____mg
- b. day to be seen by GP for monitoring of INR levels _____

_____ 2. Minimum of 4 weeks warfarin therapy post DC cardioversion
- a. dose _____mg
- b. day to be seen by GP for monitoring of INR levels _____

Follow-up Care

_____1. Consult Cardiology
_____2. Admit for further investigations and/or stabilization
_____3. Book appointment for outpatient TEE/2D echo
DATE: _____
_____4. Book appointment for outpatient DC Cardioversion if necessary
DATE: _____
_____5. Discharge ensuring follow-up for monitoring of INR levels if at high risk for embolization (long standing history of CHF, HTN)

Unsuccessful DC cardioversion

Rate Control (preferably less than 90 beats/minute)

_____ 1. Heart Function Preserved (> 40%) – Use only one of the following:

a. Initial (IV)dose:
- _____ Diltiazem (0.25 mg/kg) IV
- _____ Metoprolol (2.5 – 5.0 mg; up to 3 doses) IV
- _____ Other medications:

a. Maintenance (Oral) dose:
- _____ Diltiazem 120-360 mg daily in divided doses
- _____ Metoprolol 25-100mg BID
- _____ Other medications:

_____ 2. Heart Function Impaired (< 40%) – Use only one of the following:

a. Initial (IV) dose:
- _____ Digoxin (0.5 mg; followed by 0.25 mg q6h x 2 prn) IV
- _____ Diltiazem (0.25 mg/kg) IV
- _____ Other medications:

b. Maintenance (Oral) dose:
- _____ Amiodarone 800mg daily for one week; 600 mg daily for one week; 400 mg daily for 4-6 weeks; usual maintenance dose 200 mg daily
- _____ Digoxin 0.125-0.375 mg daily
- _____ Diltiazem 120-360 mg daily in divided doses
- _____ Other medications:

Further diagnostics if warranted

_____1. Blood work:
- _____ STAT: CBC differential, sodium, potassium, chloride, CO_2, creatinine, blood urea, magnesium, and calcium
- _____ STAT: PT (INR)/PTT
- _____ Consider CK, CKMB, and Troponin if suspecting ischemia
- _____ Consider TSH
- _____ Consider toxicology screen if suspecting substance abuse

_____2. Chest x-ray

Oral Anticoagulation

_____ 1. Minimum of 3 weeks warfarin therapy pre outpatient DC cardioversion if necessary
  a. dose _____mg
  b. day to be seen by GP for monitoring of INR levels _____
_____ 2. Minimum of 4 weeks warfarin therapy post DC cardioversion
  a. dose _____mg
  b. day to be seen by GP for monitoring of INR levels _____

Follow-up Care

_____1. Consult Cardiology
_____2. Admit for further investigations and/or stabilization
_____3. Book appointment for outpatient TEE/2D echo
  DATE: _____
_____4. Book appointment for outpatient DC Cardioversion if necessary
  DATE: _____
_____5. Discharge ensuring follow-up for monitoring of INR levels if at high risk for embolization (long standing history of CHF, HTN)

STABLE

Diagnostic Workup

_____1. 12 lead ECG
_____2. Continuous telemetry monitoring
_____3. VS including O_2 @ ___L by np prn; titrate to saturation > 90%
_____4. Initiate 1 IV site (0.9% NS @ _____cc/hr)
_____5. Blood work:
  _____ STAT: CBC differential, sodium, potassium, chloride, CO_2, creatinine, blood urea, magnesium, and calcium
  _____ STAT: PT (INR)/PTT
  _____ Consider CK, CKMB, and Troponin if suspecting ischemia
  _____ Consider TSH
  _____ Consider toxicology screen if suspecting substance abuse
_____6. Chest x-ray
_____7. NPO in preparation for DC Cardioversion if warranted
_____8. Weight _____ kg; Height _____cm

Delay DC cardioversion (if at high risk for embolization)

_____ 1. Anticoagulation Oral (unless contraindicated)
_____ 2. Consider TEE or 2D echo echocardiography for individuals with significant predisposing risk factors (i.e., Mitral Regurgitation, Hypertension, Heart Failure increasing their risk for a cerebrovascular infarction) who become severely symptomatic during treatment, thus requiring emergent DC cardioversion

Rate Control (preferably less than 90 beats/minute)

_____ 1. Heart Function Preserved (> 40%) – Use only one of the following:
  a. Initial (IV) dose:
    _____ Diltiazem (0.25 mg/kg) IV
    _____ Metoprolol (2.5 – 5.0 mg; up to 3 doses) IV
    _____ Other medications:

  b. Maintenance (Oral) dose:
    _____ Diltiazem 120-360 mg daily in divided doses
    _____ Metoprolol 25-100mg BID
    _____ Other medications:

_____ 2. Heart Function Impaired (< 40%) – Use only one of the following:
  a. Initial (IV) dose:
    _____ Digoxin (0.5 mg; followed by 0.25 mg q6h x 2 prn) IV
    _____ Diltiazem (0.25 mg/kg) IV
    _____ Other medications:

b. Maintenance (Oral) dose:
_____ Amiodarone 800mg daily for one week; 600 mg daily for one week; 400 mg daily for 4-6 weeks; usual maintenance dose 200 mg daily
_____ Digoxin 0.125-0.375 mg daily
_____ Diltiazem 120-360 mg daily in divided doses
_____ Other medications:

Oral Anticoagulation

_____ 1. Minimum of 3 weeks warfarin therapy pre outpatient DC cardioversion
a. dose _____mg
b. day to be seen by GP for monitoring of INR levels _____
_____ 2. Minimum of 4 weeks warfarin therapy post DC cardioversion
a. dose _____mg
b. day to be seen by GP for monitoring of INR levels _____

Follow-up Care

_____1. Consult Cardiology
_____2. Admit for further investigations and/or stabilization
_____3. Book appointment for outpatient TEE/2D echo
DATE: _____
_____4. Book appointment for outpatient DC Cardioversion if necessary
DATE: _____
_____5. Discharge ensuring follow-up for monitoring of INR levels if at high risk for embolization (long standing history of CHF, HTN)

Appendix B

Atrial Fibrillation Critical Pathway Questionnaire

August 1, 2003

Dear Colleague:

As you have been aware, The Critical Pathway for Atrial Fibrillation has been implemented at the Grey Nuns Community Hospital Emergency Department. Enclosed is a questionnaire seeking your feedback following the use of The Critical Pathway for Atrial Fibrillation. Your responses will be completely anonymous.

Please return the completed questionnaire by September 1, 2003 in the self-addressed envelope provided.

Thank you in advance for participating in this project. If you have any questions or concerns, please feel free to contact me at 497-4245.

Yours sincerely,

Colleen Maykut, RN, BScN, MN (candidate)

Faculty of Nursing, University of Alberta

Atrial Fibrillation Critical Pathway Questionnaire

T he purpose of this questionnaire is to determine the satisfaction healthcare professionals have with the implementation of The Critical Pathway for Atrial Fibrillation. The publication of the information obtained from this questionnaire will in no way identify your personal responses.

Instructions: Please rank the following questions using the following descriptors:

1 = strongly disagree, 2 = disagree, 3 = neutral/no opinion, 4 = agree, 5 = strongly agree.

1. Was The Critical Pathway for Atrial Fibrillation simple to initiate?

1 2 3 4 5

Comments:

2. Is The Critical Pathway for Atrial Fibrillation clear to follow?

1 2 3 4 5

Comments:

3. Is The Critical Pathway for Atrial Fibrillation complete in the treatments suggested?

1 2 3 4 5

Comments:

4. Is The Critical Pathway for Atrial Fibrillation useful to your practice?

1 2 3 4 5

Comments:

5. Is The Critical Pathway for Atrial Fibrillation helpful in providing care?

1 2 3 4 5

Comments:

6. Did you use The Critical Pathway for Atrial Fibrillation?

Yes _____ No _____

If NO, please comment:

Additional Comments (please feel free to use the back):

Thank you for completing this questionnaire!

Appendix C

American College of Cardiology & American Heart Association Recommendations

Class I	Conditions for which there is evidence for and/or general agreement that the procedure or treatment is useful and effective.
Class II	Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment. **Class IIa** The weight of evidence of opinion is in favour of the procedure or treatment. **Class IIb** Usefulness/efficacy is less well established by evidence or opinion.
Class III	Conditions for which there is evidence and/or general agreement that the procedure or treatment is not useful/effective and in some cases can be harmful.

Appendix D

Data Collection Tool

Group A B

Hospital ID#: _____ Study ID#: _____

1. Demographics

Age:____ Gender: M F BMI: _____

Co-morbidities: **Risk Factors:**

___HTN___Valvular Heart Disease
___CAD___MI___CHF___CABG___Family Hx
___Recent surgery___Thyroid disease
___Diabetes___Acute pulmonary disease
___Substance abuse___Infection
___Acid-Base or electrolyte imbalances

Heart Function: Preserved/Impaired (EF____%)

Medications (Including OTC and Herbal)

Acute Alcohol consumption yes ____ no ____

First occurrence of AF: yes no? If no # of times ____

2. ED Initial Assessment

Time of arrival at ED _____ Time seen by physician_____

Length of stay in ED _____minutes

Baseline Temp _____ HR_____ BP_____ O_2Sat_____

Length of time in AF _____

Symptom presentation:

3. Initiation of Critical Pathway for AF

Time to initiate protocol _____minutes

If not initiated, rationale:

4. Check ordered treatment and diagnostic interventions:

_____1. Continuous telemetry monitoring
_____2. VS including O_2 @ ___L by np prn; titrate to saturation > 90%
_____3. 12 lead ECG
_____4. Initiate 2 IV sites (0.9% NS @ _____cc/hr and a NS lock)
_____5. Blood work:
- _____ STAT: CBC differential, sodium, potassium, chloride, CO_2, creatinine, blood urea, magnesium, and calcium
- _____ STAT: PT (INR)/PTT
- _____ Consider CK, CKMB, and Troponin if suspecting ischemia
- _____ Consider TSH
- _____ Consider toxicology screen if suspecting substance abuse

_____6. Chest x-ray
_____7. NPO in preparation for DC Cardioversion if applicable
_____8. Weight _____ kg; Height ___cm
_____9. Consider rate control and anticoagulation in those individuals presenting with onset of AF<48 hours prior to cardioversion with a potential risk for embolization (i.e. long standing history of CHF, HTN)
____10. Other

AFIB <48 Hours > 48 Hours

* If **AMIODARONE** was not used for both **RATE CONTROL** and **PHARMACOLOGICAL CONVERSION** in AF < 48 hours, please give rationale:

5. Rate Control

A. Identify applicable drugs with dosages:

___ Diltiazem IV _____dose

___ Metoprolol IV _____dose

___ Verapamil IV _____dose

___ Digoxin IV _____dose

Other, please list:

B. Rate control achieved yes no _____beats/min

6. Convert Rhythm

A. DC CARDIOVERSON

a. Ventricular rate _____beats/min

b. Signs and symptoms related to tachycardia resulting in choosing DC cardioversion please list:

c. Conscious sedation _____dose _____mg

d. Use of antiarrhythmic drug prior to cardioversion yes/no

List drug and dose if applicable: _____

e. # of joules and frequency

f. Able to convert yes/no

g. Remain in SR yes/no

B. PHARMACOLOGICAL CONVERSION (check applicable drugs with dosages):

___ Flecainide po _____dose

___ Procainamide IV _____dose

___ Propafenone po _____dose

7. Anticoagulation

A. Prior to emergent DC cardioversion yes no

___ Unfractionated heparin

___ Bolus _____dose

___ Infusion _____dose

___ Low Molecular Weight Heparin yes no

___ Dalteparin _____dose

___ Enoxaparin _____dose

B. Pre Out-patient cardioversion

___ Minimum of 3 weeks

C. Post Out-patient cardioversion

___ Minimum of 4 weeks

8. List identifiable precipitants: non-cardiovascular or cardiovascular

A.

B. Treated yes no

9. Follow-up Care

_____1. Consult Cardiology

_____2. Admit for further investigations and/or stabilization

_____3. Book appointment for outpatient TEE/2D echo
DATE: _____

_____4. Book appointment for outpatient DC Cardioversion
DATE: _____

_____5. Discharge ensuring follow-up for monitoring of INR levels

_____6. Discharge with no instructions